3
EAR-GUM

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VOL: III

EARTH BURIAL TO GUMMI INDICUM

WILLIAM GREEN & SONS
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SCOTT CALDER

EDITORIAL NOTE

THIS volume (the third) carries the subject-matter of the *Encyclopedia and Dictionary of Medicine* from EARTH to GUMMI INDICUM, and it completes nearly one-third of the whole work.

There are in this section 1097 subject-headings, distributed in the following manner. There are sixty articles of more than 1000 words in length; these include contributions to the subjects of *Eclampsia, Ectopic Gestation, Eczema, Injuries and Diseases of the Elbow-Joint, Electricity, Electrolysis, Emetics, Enemata, Enteroptosis, Enzymes, Ephemeral Fever, Epidemiology, Epilepsy, Epiphyses, Epistaxis, Equilibrium, Erysipelas, Erythema, Expectoration, Examination of the Eye and Diseases of the Eyeball and Eyelids, Facial Hemiatrophy, Facial Paralysis and Facial Spasm, Fæces, Fallopian Tubes, Fascia, Favus, Affections of the Fifth Nerve, Filariasis, Fingers, First Aid, Examination of Pathological Fluids, Development of the Fœtus and Ovum, Food, Foot and Mouth Disease, Fractures, Furunculus Orientalis, Diseases of the Gall-Bladder and Bile Ducts, Galvanic Cautery, Ganglion, Gangrene, Gastro-Intestinal Disorders of Infancy, General Paralysis, The Female Organs of Generation and their Arrested Development, Glanders, Glandular Fever, Glaucoma, Glycosuria, Gonorrhœal Infection, Gout,* and *The Groin.* These articles appeared originally in the *Encyclopædia Medica*; in some instances, in which the progress of Medicine rendered it advisable, they have been brought up to date either by thorough revision (*e.g.* in the case of the article dealing with the *Affections of the Gall-Bladder and Bile Ducts*) or by the insertion of supplementary paragraphs or articles (*e.g.* in connection with the subjects of *Eclampsia* and *Ectopic Gestation*). Entirely new articles on *Embryology,* on *Ferrum,* on the *Fourth Disease,* and on *Erythema Infectiosum* have been added.

There are over one hundred articles of less than 1000 words, but of more than 10 lines (80 to 900 words) in length; these deal with such widely different subjects as *Earthquakes* in their relation to Epidemics, the "*Ecchymotic Mask,*" *Eclecticism, Ecstasy, Ecstrophy, Ectopia, Eddyism, Elaterium, Embolism, Embolus, Emigration of Leucocytes, Emmenagogues, Surgical Emphysema, Hours of Employment, Eosinophilia, Epispadias, Ergot, Erythromelalgia, Medical Ethics, Ethnology, Eucalyptus, Eugenics, Exalgin, Expectorants, Facial Hemihypertrophy, Facies, Fat Necrosis, Fatty Degeneration, Fecundity, Ferrometer, Filix Mas, Fomentation, Formaldehyde, Formic Acid, Fragilitas Ossium, Galbanum, Galls, Gamboge, Garden Cities, Garrulitas Vaginæ, Gelatin, Gelsemii Radix, Germinal Pathology, Ginger, Glosso-Pharyngeal Nerve, Glycerin, Glycogen Reaction,* and *Grindelia.*

Finally, there are 935 short articles and headings, varying in length from 10 lines

to a few words. Many of these are definitions of medical terms, new and old, while some are cross-references which serve almost as definitions. Among the matters which these short articles define or deal with may be named *Easton's Syrup, Ebstein's Theory, Echo Sign, Eck's Fistula, Écouvillonage, Ectopagus, Effusion, Egyptian Anæmia, Elecampane, Electron, Elixir, Embalming, Embryocardia, Embryoma, Empirical, Endothelioma, End Products, Enentery, Enterokinase, Eparsalgia, Epidermolysis, Epigenesis, Epiphora, Episiotomy, Esters, Euhemerism, Euthanasia, Ewald's Test Meal, Expectation of Life, Exploratory Operations, Eyestrain, Felo-de-se, Fibrin, Ficus, Fillet, Finsen Light, Fitz-James Colony, Fleabane, Flint's Murmur, "Flush Area," Fomites, Foveola Coccygea, Four-footed Progression, Fränkel's Posture Test, Free Martin, Fryer's Destructor, Fulmar Oil, Funnel Breast, Gait, Galenism, Galton's Law, Gamma Rays, Ganser's Syndrome, Gasserectomy, Gavage, Genetous Idiocy, Genupectoral Position, Gerber's Process, Germ, Ginseng, Glabella, Glue-Making, Glycerophosphates, Goose-Cough, Goundu, Gruebler's Tumour,* etc.

Now that these three volumes have appeared, it will be evident to the reader that the cross-references are of great value in opening up, as it were, the contents of the *Encyclopedia* to his use, and in facilitating immediate reference to all the aspects of any given subject. This will be increasingly evident as volume after volume leaves the press.

J. W. BALLANTYNE.

JANUARY 1907.

CONTENTS

THE CHIEF ARTICLES IN VOLUME III., WITH THEIR AUTHORS

LIST OF ILLUSTRATIONS

ENCYCLOPEDIA AND DICTIONARY OF MEDICINE AND SURGERY

Earth Burial.—The method of disposal of the dead commonly employed in this country. *See* Cemeteries; Burial Places; etc.

Earth Closet.—A dry method of treating the fæces, by which earth instead of water is added to the deposit to act as a deodorant and absorbent of the excreta; dry sifted earth (loam or brick-earth) is used, and about one pound and a half is needed each time; Moule's system. *See* Sewage and Drainage (*Conservancy System*).

Earth Eating.—The form of perverted appetite in which earth, cinders, and the like are eaten. *See* Appetite (Perversion); Hysteria; Pica; Pregnancy, Management ("Longings").

Earthnut Oil.—Arachis oil (*Oleum Arachis*), ground-nut oil, or pea-nut oil, is the expressed oil of the seeds of Arachis hypogæa; and it is used in India and the Colonies instead of olive oil; it is included in the Indian and Colonial Addendum to the British Pharmacopœia of 1898.

Earthquakes.—The scientific investigation of earthquakes and earth tremors (seismology) which is now being carried on in various parts of the world will, no doubt, ere long enable us to trace the effect (if any) which exists between earthquakes and epidemics; it would seem that a destructive earthquake may, by throwing out of order the system of drainage in a town, by disturbing the water-supply, and by submitting the inhabitants to privation, be the indirect cause of epidemic outbreaks of disease. *See* Epidemiology.

Earwigs. *See* Nose, Foreign Bodies (*Parasites, Entomozoaria*).

Eastbourne. *See* Therapeutics, Health Resorts (English).

Easton's Syrup.—The syrup of the three phosphates (*Syrupus Ferri Phosphatis cum Quinina et Strychnina*), given in doses of $\frac{1}{2}$ to 1 fl. dr.; a favourite tonic; it is to be remembered that each fluid drachm contains $\frac{1}{32}$ of a grain of strychnine. *See* Iron; etc.

Eating. *See* Food; Physiology, Food and Digestion; etc.

Eau de Cologne. *See* Alcohol (*Varieties of Alcoholism*); Morphinomania and Allied Drug Habits (*Eau de Cologne*).

Eau de Javelle.—A disinfectant solution, also known as Labarraque's liquor; it is the *Liquor Sodæ Chlorinatæ* of the *Pharmacopœia*.

Eau de Vie.—Brandy. *See* Alcohol (*Spirits*).

Eaux Bonnes. *See* Balneology (*France, Sulphur Waters*).

Eaux Chaudes. *See* Balneology (*France, Sulphur Waters*).

Eberth's Bacillus. *See* Typhoid Fever (*Etiology, Characters of Bacillus typhosus*).

Ebstein's Method. *See* Obesity (*Etiology, Treatment*).—A dietetic method of treating obesity in which fat is allowed in the dietary.

Ebstein's Theory.—The theory that necrotic changes in the tissues are the primary cause of gout, the necrosis being due to the presence of dissolved urates in the body-fluids. *See* Gout (*Gout in Lower Animals*).

Ebullition.—The process of boiling (*e.g.* in Pharmacy); also, formerly, the supposed state of agitation in the blood or "humours" giving rise to inflammation and fever.

Eburnation.—An ivory-like condition of osseous tissue, due to excessive deposition of calcareous material; also, ossification of articular cartilage. *See* RHEUMATOID ARTHRITIS.

Ecballium Elaterium.—The Squirting Cucumber, from the fruit of which the drastic purgative, elaterium, is obtained. *See* ELATERIUM.

Ecbolics.—Medicinal substances which stimulate contractions in the parturient, puerperal, and (sometimes) in the pregnant uterus; they include ergot, quinine, savin, and all powerful purgatives, and they all act also as emmenagogues; oxytocics. *See* ABORTION; EMMENAGOGUES; ERGOT; PHARMACOLOGY; etc.

Eccentricity.—A deviation or tendency to deviate from the usual or normal in habits and modes of action; oddity or irregularity in behaviour. *See* INSANITY, NATURE AND SYMPTOMS.

Ecchymosis.—An extravasation or effusion of blood, from Gr. ἐκχυμόεσθαι, to extravasate blood, into the subcutaneous or submucous or subserous tissue, causing a patch of discoloration; it cannot be removed by pressure; when the extravasated blood is sufficient in amount to cause a swelling, it is termed an *Ecchymoma*. *See* HYSTERIA (*Disorders of Circulation, Ecchymoses*); POST-MORTEM METHODS (*External Examination, Ante-mortem Bruising and Post-mortem Lividity*); PURPURA (*Character of the Cutaneous Hæmorrhage*); SCURVY IN ADULTS (*Clinical Features, Ecchymoses*).

"Ecchymotic Mask."—The rare result of sudden and forcible compression of the chest or abdomen, or of both; the face and neck become at once suffused, and take on a continuous violet coloration with darker spots here and there; it is believed that strong, although unconscious struggling efforts on the part of the person so crushed may aid in the production of the "mask"; recovery may take place, and when death occurs it is usually due to multiple injuries of the abdominal and thoracic organs or of the skeleton.

Eccoprosis.—Defæcation (from Gr. ἐκ, out, and κόπρος, dung); an *eccoprotic* is a mild purgative medicine, and *eccoprosiæsthesis* is the sensation of the need of evacuating the bowels.

Eccyesis.—Ectopic or extra-uterine pregnancy. *See* ECTOPIC GESTATION.

Ecdemic.—As applied to disease ecdemic means arising from a cause outside the locality, and not affecting the great bulk of the population; it is, therefore, an antonym of endemic and epidemic.

Ecgonine.—An alkaloid ($C_9H_{15}NO_3 + H_2O$), obtained by the decomposition of cocaine; with benzoic anhydride it yields benzoyl-ecgonine, and, on the other hand, cocaine, long in contact with hot water, acids, or alkalies, breaks up into benzoyl-ecgonine and methyl alcohol ($C_{17}H_{21}NO_4 + H_2O = C_{16}H_{19}NO_4 + CH_3OH$); ordinary cocaine is benzoyl-methyl ecgonine. *See also* COCAINE.

Echinococcus.—The larva of Tænia Echinococcus or the Hydatid. *See* HYDATID DISEASE; *see also* BROAD LIGAMENT, DISEASES (*Echinococci*); LIVER, DISEASES (*Cysts*); PARASITES (*Cestodes*); SPLEEN, SURGERY OF (*Echinococcus Cysts*).

Echinorrhynchus.—An unsegmented worm, having a protractile proboscis (Gr. ἐχῖνος, hedgehog, and ῥύγχος, snout), occurring as an intestinal parasite in the lower animals, rarely in man; there is the *echinorrhynchus hominis*, the *e. gigas*, and the *e. moniliformis*. *See* PARASITES (*Acanthocephali*).

Echis. *See* SNAKE-BITES (*Viperidæ, Echis or "Fursa"*).

Echitamine.—An alkaloid ($C_{22}H_{28}N_2O_4$) of "Dita" (the bark of *Echites* or *Alstonia scholaris*); ditaine. *Echitenin* ($C_{20}H_{27}NO_4$) is another alkaloid from the same source; and *echicerin* ($C_{30}H_{48}O_2$), *echitein* ($C_{42}H_{70}O_2$), and *echitin* ($C_{32}H_{51}O_2$) are yet other substances. There are various species of *Echites*, which are used medicinally in S. America, the East Indies, and W. Africa.

Echolalia. *See* ECHO SPEECH.

Echopathies.—Nervous maladies in which the patient spasmodically and involuntarily repeats at once words and actions heard and seen by him; the name *echokinesis* is given to the repetition of actions.

Echo-Sign.—A speech disturbance met with in epilepsy, consisting in the repetition of a word two, three, or more times at the end or in the middle of a sentence.

Echo Speech.—The power of repeating, like a parrot, words spoken without understanding the same; echolalia or echophrasia, *See* APHASIA (*Clinical Features, Word-deafness*); SLEEP, NORMAL AND MORBID (*Morbid Somnolence, Katatonia*).

Eck's Fistula.—An experimental means devised by Eck for excluding the liver from the circulation, by uniting the peripheral end of the divided portal vein with the inferior vena cava. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Urea-Formation*).

Eclampsia.

See also Convulsions, Infantile; Head-Shaking (*Spasmus Nutans*); Labour, Operations, Induction of Premature Labour (*Indications*); Labour, Operations, Forceps (*Indications, Maternal Complications*); Labour, Operations, Embryotomy (*Indications*); Liver, Diseases (*Functional Disease in Puerperal Eclampsia*); Nephritis (*Clinical Features, Chronic Nephritis, Nervous System*); Pregnancy, Physiology (*General Changes, Toxæmia*); Pregnancy, Affections and Complications (*Nervous Systems, Renal Affections, Albuminuria*); Thyroid Extract; Uræmia (*Symptoms, Convulsive Attacks*); Urine, Pathological Changes in (*Proteids, Albuminuria*).

Definition.—Eclampsia is the term applied to epileptiform attacks occurring in pregnant or puerperal women, which are the manifestations of a cerebral intoxication or over-activity arising as an indirect result of the pregnancy. By thus defining eclampsia we wish to make it clear that it is not a term to be applied to one particular form of convulsive attack which occurs during pregnancy, but is rather to be applied to any form of convulsive attack which may occur from any cause, provided that cause is primarily due to the effects of pregnancy on the organism of the patient.

Frequency.—The frequency of eclampsia is rather a difficult matter to ascertain. If the statistics of lying-in hospitals are followed the percentage which is thus obtained will be too high, as there will always be found a relatively greater number of primiparæ than of multiparæ in hospitals, and also as a patient is more likely to seek the aid of a hospital if she feels herself seriously ill than if she is expecting her confinement in her ordinary health. The statistics of various British and continental hospitals and clinics give the following figures:—Amongst 227,000 patients there were 635 cases of convulsions, *i.e.* a proportion of 1 case in 357·48. All these cases may not have been eclampsia, but the proportion very closely corresponds to that at the Rotunda Hospital, where amongst 11,958 patients there were 36 cases of eclampsia, *i.e.* 1 case in 332·16.

Pathological Anatomy.—At the autopsies of patients who have died of eclampsia a tolerably uniform series of pathological conditions are met with in the various organs. The organ most constantly affected, and the one whose condition is most closely connected clinically with the symptoms of the case, is the kidney. The next most constantly affected organ is the brain, and after it the liver. There is, however, no one lesion which has come to be definitely regarded in the light of a primary lesion. If the various organs are examined one by one the following changes are found:—

Liver.—The surface of the liver is scattered over with patches caused by ecchymosed blood. Some of these are the size of a pin's head, while others may be half the size of the palm of the hand. On section the colour of the liver substance is more yellow than usual, owing to various lesions of the epithelium (Pilliet). Hæmorrhages, resembling the above subcapsular hæmorrhages, are found scattered round the portal interspaces. Under the microscope they are seen in three different stages. In the first stage they consist of a circular area of dilated intralobular capillaries, situated close to a portal space, and about the size of a grape-stone. In the second stage these areas of engorgement have increased in size. Round their periphery there is a ring of dilated capillaries, while the centre has become necrotic, and consists of a mass of dead liver cells, blood corpuscles, vessels, and fibrin. In the third stage the areas have still further increased, and in places where they were near one another they have coalesced. In this way islands of necrosis are formed surrounded by a small-celled exudation. From these areas emboli of liver cells (Jürgens)—of fat (Virchow)—may be carried to other organs.

With regard to the origin of the hæmorrhages which produce these necrotic areas there is a wide difference of opinion, as we shall see in discussing the various theories of eclampsia. Here it is sufficient to say that they may occur in one of several ways: first, as the result of the bringing to the liver, in the blood, of some toxic substance—chemical or bacterial in origin—which destroys the liver cells; secondly, as the result of embolic infarction of the liver, the emboli coming from the placenta, or being formed by the action of some coagulation-producing ferment on the blood; and, thirdly, as the result of the rupture of small blood-vessels during an eclamptic fit. Similar areas are found in the spleen, kidney, pancreas, brain, and lungs.

Kidneys.—In from 90 to 95 per cent of cases the kidneys are affected, most commonly by the condition known as pregnancy kidney. According to Leyden this is not a true nephritis, but rather the result of simple anæmia, and is characterised by a fatty infiltration of the renal epithelium, especially of the convoluted tubes. The cause of this anæmia is attributed by Dührssen, Spiegelberg, and others to spasm of the renal arteries, due to their reflex irritation by stimuli from the genital tract. Such stimuli are furnished by the contractions or great distension of the pregnant uterus, by the entrance of the head into the pelvis, and by the onset of

labour. Volhard attributes renal anæmia to the blocking of the nutrient vessels by emboli caused by the action of some coagulation-producing ferment upon the blood. Much more rarely the lesions of true chronic nephritis are found associated with eclampsia, and in a very small proportion of cases the renal changes can be attributed to the effects of obstructive suppression of the urine, due to pressure on the ureters (Halbertsma).

The same areas of necrosis as are found in the liver are also to be sometimes found in the kidneys, and can be attributed to like causes.

Spleen.—The spleen is enlarged, congested, and diffluent. Subcapsular hæmorrhages and similar areas of necrosis as are found in the liver are sometimes present.

Pancreas.—The pancreas also presents areas of necrosis, and may be markedly anæmic.

Brain.—The brain is sometimes hyperæmic, sometimes anæmic. There is usually marked œdema, leading to consequent flattening of the convolutions, and minute hæmorrhages may occur in various parts.

Lungs.—The lungs are congested, especially at their bases. There are also sub-pleural ecchymoses, and emboli with necrotic areas are found as in other organs.

Fœtus and Placenta.—Somewhat similar conditions have been found in the liver and kidneys of the fœtus as are described as occurring in the mother. The placenta is frequently the subject of white infarction, and it is suggested that from these areas placental giant-cells may pass into the maternal blood and cause coagulation, as well as acting as emboli.

ÆTIOLOGY.—The number of theories which have been advanced to account for the occurrence of eclampsia is sufficient evidence of the amount of uncertainty with which the pathology of the disease is surrounded. The most important of these are the following:—

Frerichs' Theory.—Frerichs' theory, that eclampsia is due to the presence in the blood of urea, or of carbonate of ammonia formed from urea under the influence of some fermentative process, is untenable for several reasons. There has not been found any storage of urea in the liver or muscles in the case of patients who have died of eclampsia; nor, in the case of those who recover, is there any increased quantity excreted. Furthermore, urea has been injected into the blood without causing convulsions. Bouchard even attributes a diuretic effect to it, and under his advice Pinard employed it, as a hypodermic injection, in 1887, in the case of anuric eclamptics. The carbonate of ammonium theory has been overthrown by Bernard, who demonstrated the fact that it was present in the same proportion in the blood of a healthy man as in that of an eclamptic.

Stumpf's Theory.—Stumpf's theory, that the fits are due to the circulation in the blood of some poison produced by an abnormal decomposition in either mother or child, has received a certain amount of revived support of late. Stumpf considers that, "under abnormal processes of decomposition, a substance free from nitrogen, toxic in its action, perhaps acetone, or a body resembling it which reacts to the same tests, may be formed. That this body produces by its excretion an irritation of the kidneys which may eventually lead to nephritis, has a destructive effect upon the colouring matter of the blood, greatly alters the activity of the liver cells, causes sugar to appear in the urine, and produces the destruction of the parenchyma of the liver leading to acute yellow atrophy of the organ with the formation of tyrosin and leucin, and induces coma and convulsions from an irritation of the brain." Fehling has quite recently lent support to this theory. According to him it may be that the metabolism of the fœtus and the transference of the final products into the maternal circulation are of more importance than has hitherto been supposed. The nephritis of pregnancy is, he thinks, most probably not the cause of eclampsia, but the first sign of intoxication, of which eclampsia, if it supervenes, may be the second. The fact that the fœtus almost always dies in these cases, the predisposition to eclampsia in the case of multiple pregnancy, and the improvement in the prognosis for the mother which the death of the fœtus affords, are all in favour of the supposition of the cause of eclampsia being produced in the fœtus. Schmorl ascribes eclampsia to an intoxication by coagulation-producing ferments which originate in the placenta.

The Urinæmic Theory.—The theory which is perhaps the most widely adopted at present is that the fits are due to the retention of the normal urinary toxines owing to the failure of function on the part of the kidney, *i.e.* that they are a symptom of urinæmia. To such a poisoning all the constituents of the urine would contribute. Coincidently with the onset of the premonitory symptoms of eclampsia the urine has been found to diminish in toxicity. It also diminishes in amount, so that there must be a consequent retention of the normal urinary toxines in the body. Coincidently with the recovery of the patient the toxicity of the urine increases, as also does the total amount of urine passed. The fact that eclampsia so frequently occurs in patients suffering from renal disease, and that it rarely or never occurs when this condition has been so treated that urinary suppression does not occur, are strong points in favour of this theory; while, on the other hand, the latter furnishes no explanation of the morbid appearances which are met with in the liver. Neither does it account for those cases—about 5 per cent of the entire number—in which there is no evidence of renal disease. The necrotic areas in the liver may, however, be the result of increased

blood-pressure occurring during the attacks, while those cases in which there is no renal disease can be explained by the neurotic theory.

Bouchard's Theory.—Bouchard's theory—the so-called auto-intoxication theory—though that term will also apply to the last—attributes eclampsia not only to a failure of function of the kidneys, but also of the liver. As a result intoxication occurs, not only from urinary extractives, but also from biliary substances which remain in the blood, and from ptomaines which are no longer destroyed in the liver. Auvard and Rivière add to this theory the effects of the failure of elimination in the skin and lungs, while Bouffe de Saint-Blaise considers the hæmorrhagic infarcts of the liver as *the* pathognomonic lesion of eclampsia, to which even the convulsions themselves are of secondary importance. The cause of this lesion, he thinks, may be found in some chemical or septic toxine which is formed in the intestine, and is brought to the liver by the blood. There can be no doubt that hepatic as well as renal incompetence plays a certain part in the pathology of eclampsia, but whether this incompetence is primary or secondary is extremely doubtful. All that can be said on this point is that there is far more reason for considering renal incompetence primary than there is for considering hepatic incompetence so. And if a doubt exists as to the primary nature of the former, it *a fortiori* exists as to the latter.

The Bacterial Theory.—The bacterial theory of eclampsia has never made much advance, although it has had from time to time strong supporters. Herrgott attributes some cases to such a cause, while Stroganoff strongly upholds the view that eclampsia is a contagious disease.

The objections to a bacterial theory are—

(1) That eclampsia is never an epidemic disease.
(2) That it is more common amongst primiparæ than amongst multiparæ.
(3) That no bacterium constant in its presence or capable of reproducing the disease has been isolated.

The Neurotic Theory.—The neurotic theory attributes eclampsia to a heightened irritability of the nerve-centres, or to excessively strong stimuli from the uterus (eclampsia reflectorica). This theory—to which support is given by instances of eclampsia which show the influence of heredity or of a neurotic disposition—will explain those cases in which no evidence of renal disease can be found. Ribemont-Dessaignes and Guéniot bring it to the assistance of the renal auto-intoxication theory as furnishing the necessary predisposing factor by the concurrence of which poisoning by urinary extractives can cause the onset of eclamptic attacks.

In addition to these theories there are certain facts which are known to predispose to eclampsia. These are:—

(1) Acute and chronic disease of the kidney, especially that form known as pregnancy kidney.
(2) Obstructed delivery.
(3) A neurotic temperament, especially if hereditary.
(4) Old and very young primiparæ.
(5) Long retention of the excretions.
(6) Multiple pregnancy.

The foregoing are the principal theories on the causation of eclampsia and the known predisposing facts. Taking into consideration the great number of the former which have been brought forward, and for the majority of which there is some support, it seems most rational in the present state of our knowledge to consider eclampsia, not as the result of one definite condition, be it of the liver, or of the kidney, or of the higher centres, but rather as the result of the undue stimulation of the nerve-centres by toxic substances—the direct or indirect result of the pregnancy—circulating in the blood, or of peripheral irritation from the genital tract. In each patient the nature of the toxine or of the peripheral irritant may differ, and with it the special symptoms of the case, but in each the pathognomonic symptom will be the occurrence of convulsive attacks. Looking at the origin of eclampsia in this light we find that its causes can be classified in general terms in the following manner:—

I. The poisoning of the nerve-centres by toxic substances circulating in the blood owing to—

(1) The accumulation of normal toxines in the blood from failure of the renal, hepatic, or intestinal eliminatory functions, due to pre-existing disease of these organs.
(2) The excessive formation of normal toxines, or the formation of abnormal toxines, either in the mother or the fœtus, which in their process of excretion through the kidneys cause nephritis, and hence a diminished renal function, and hence a further increased amount of toxines in the blood.

II. The over-activity of the nerve-centres, due to—

(1) Their over-excitability to normal stimuli, as in the case of hysterical patients or epileptics.
(2) Their over-irritation by excessive stimuli, as in the case of obstructed labour, very painful labour pains, very old or very young primiparæ.

Symptoms.—The symptoms of eclampsia must be considered under two heads:—

(1) Prodromal.
(2) Actual.

(1) *Prodromal Symptoms.* — The first pro-

dromal symptom of eclampsia may be said to show itself the moment a pregnant woman passes urine containing albumin, if previously her urine was healthy. In this connection the following rule may be given. It is *advisable* to examine the urine of every pregnant woman during the sixth and seventh months, and to ascertain the amount passed in twenty-four hours. It is *absolutely necessary* to do so if from her history or appearance we have any grounds for supposing that she may be suffering from Bright's disease. The more immediate prodromal symptoms of eclampsia occur a short time before the onset of the fits in the great majority of cases. The earliest possible recognition of these symptoms is a matter of necessity, as by so doing it is possible in many cases to completely ward off the threatened attack. They consist in complete or partial, temporary or permanent, loss of vision, flashes of light before the eyes, vertigo, headache, drowsiness, mental depression, nausea, constipation, and epigastric pain. Coincidently with the foregoing the amount of urine passed is markedly diminished, and the amount of albumin in it increased.

(2) *Actual Symptoms.*—The actual symptoms commence with the onset of the fits. Eclampsia rarely or never is met with before the sixth month. It most commonly occurs between the eighth month and full term, and sometimes starts during the puerperium. A fit lasts from one to one and a half minutes, and consists of three stages—a preliminary stage, a tonic stage, and a clonic stage, followed by a varying period of coma. The preliminary stage lasts from a half to one minute. It consists of various convulsive movements of the head and facial muscles. The eyelids twitch vigorously, the eyeballs are deviated to the left and upwards, the nostrils quiver, and spasms of the muscles of respiration occur. The tonic stage then commences and lasts from fifteen to twenty seconds. The patient becomes rigid, the head thrown backwards and to the left, the trunk in a position of opisthotonos. Respiration is arrested, the jaws are tightly clenched, and the tongue, which was protruded in the preliminary stage, may be violently bitten. The clonic stage follows, and lasts a varying period, the tonic spasms passing off gradually, and being replaced by sharp rhythmical movements—the patient "works." Finally, the clonic movements cease, respiration returns, and the patient lies in a condition of deep coma. The duration of the coma varies according to the number of fits which the patient has had. At first it only lasts for a few minutes, but as the number of the fits increase it lasts in the intervals between them. The number of fits vary greatly. The patient may only have one, while on the other hand as many as a hundred have occurred. They may pass off entirely for a time, and then recur. In a severe case they follow one another at ever shortening intervals. In such a case the heart's action soon becomes affected, and is frequent and weak, and finally intermittent. The lungs are also involved, and become congested, partly as a result of the failure of the heart, and partly from the irritation caused by the entrance of particles of food and mucus—deglutition pneumonia. The temperature, which at first was normal, gradually rises as the fits recur, and may finally reach a height of 104° F. Total or partial loss of vision is also of frequent occurrence. There is almost complete suppression of urine and constipation.

Diagnosis.—The foregoing description of an eclamptic attack is the description of a typical case. It must be borne in mind that the greatest divergence from this type may be met with, and that the attack may assume the most atypical forms. For this reason too much reliance must not be placed on the form of the convulsion in making a diagnosis of the nature of the case. More information will be obtained by studying the prodromata, the history of the case, and the attendant symptoms.

Eclampsia must be distinguished from epilepsy, hysteria, drunken delirium and coma, and the coma and convulsions of meningeal and cerebral disease. As a general rule, it may be stated that every form of convulsion in a pregnant woman who is suffering from renal disease should be regarded as eclamptic in origin until the reverse is proved. Epilepsy may be recognised by the history of former attacks, by the absence of the usual eclamptic prodromata, by the initial epileptic aura, by the sharp onset of the convulsive seizure, and by the usually complete absence of all renal symptoms.

Hysteria is recognised by the extreme irregularity of the convulsion, by the absence of respiratory spasm and of all actions which would hurt the patient, the absence or loss of consciousness, and the passage of large quantities of pale urine.

Alcoholic coma and delirium may be suspected from the history of the case and the spirituous odour of the breath. It can be definitely recognised, as it gradually passes off and does not recur. The urine also is probably free from albumin.

The coma and convulsions of meningeal and cerebral disease may be indistinguishable from those of eclampsia if the history of the onset of the case cannot be obtained. It must not be forgotten that the two conditions may coexist, as cerebral hæmorrhage occasionally occurs in the course of eclampsia.

Complications.—The principal complications of eclampsia, if indeed they can be considered as such, and not rather as integral parts of the disease, are failure of the heart and consequent œdema of the lungs. Cerebral hæmorrhage may occur from the rupture of a vessel during

a fit, or even after the fits have ceased. Septic pneumonia may result from the inspiration of foreign bodies into the lungs.

TREATMENT. — The treatment of eclampsia must be considered under two heads :—

I. Prophylactic treatment.
II. Curative treatment.

I. *Prophylactic Treatment.* — Prophylactic treatment must be adopted in the case of every pregnant woman who has persistent albuminuria, especially if the urine also contains tube-casts. Such treatment must be carried out still more rigorously if any of the other prodromata of eclampsia appear. The importance attributed to prophylaxis will be shown by the following opinions :—"When a patient suffering from albuminuria has been on milk diet for a week, she almost to a certainty escapes eclampsia" (Tarnier). "Eclampsia occurs almost exclusively in women whose urine has not been examined during pregnancy" (Ribemont - Dessaignes). "The author has never yet seen a case of eclampsia occur amongst the numerous cases of kidney of pregnancy where this method (*i.e.* prophylactic treatment) has been adopted during pregnancy (Dührssen).

It is practically impossible, and it is rarely necessary, to enforce an absolute milk diet from the date at which renal disease is first recognised, *i.e.* about the sixth month, to the end of pregnancy. It will usually suffice if milk and other fluids are made to take a great share in the dietary. In addition fish, white meat, eggs, and vegetables may sometimes be allowed. If milk diet is not an absolute essential, the due regulation of the eliminatory functions of the body is. The bowels must be freely moved each day, the skin must be encouraged to act by frequent warm baths, and the amount of urine passed daily must be noted. The dietary of the patient and the daily amount of urine should be in direct proportion to one another. The freer the action of the kidneys the more liberal may be the dietary. The moment the former show any signs of failure the latter must be reduced to milk alone, to be cautiously made more varied as the renal action improves.

If the urine diminishes to a marked extent, and any of the prodromal symptoms of eclampsia appear, a hydragogue purgative must be at once administered. At the same time, to increase the action of the skin, hot baths and wet packs must be ordered, and the patient kept wrapped in blankets. A suitable purgative to administer in these cases consists of calomel 10 grs., combined with pulv. jalapæ co. 1 drachm, and followed, if necessary, as is sometimes the case, by an enema at the end of six hours.

II. *Curative Treatment.*—The curative treatment of eclampsia is directed in the main towards two principal points :—

(1) The arrest of the fits.
(2) The staving off of complications.

(1) *The Arrest of the Fits.*—The fits must be checked at the earliest possible moment, as each successive attack leaves the patient more liable to fall a victim to the complications of a failing heart and œdema of the lungs. There are three ways of attaining, or of endeavouring to attain, this end :—

(*a*) By administering sedatives.
(*b*) By removing toxic substances from the blood and tissues.
(*c*) By emptying the uterus.

(*a*) By administering sedatives.—There are two distinct lines of treatment which fall under this head—the chloral and chloroform treatment, and the morphia treatment.

The chloral and chloroform treatment consists in administering, upon the onset of an attack, 30 grs. of chloral hydrate by the rectum, and repeating it every two hours until the fits cease. Up to $3\frac{1}{2}$ drachms may be given within twenty-four hours, but not more. The inhalation of chloroform is commenced as soon as any sign of the onset of a fit is noticed, and is continued until the fit is over. The great objection to this line of treatment is that we are administering drugs which have a depressant effect upon the heart, and which consequently tend to favour the occurrence of heart failure.

The morphia line of treatment was first introduced by G. Veit. It is the treatment which the writer recommends, as he considers it superior to the chloral and chloroform treatment. It consists in the administration of half a grain of morphia hypodermically as soon as a fit occurs. A quarter of a grain is then administered every two hours until the fits cease, but not more than three grains are given in twenty-four hours. Morphia will check convulsions quite as rapidly as chloroform, as statistics show, while at the same time it has not the same depressant effect upon the heart. Its reputed bad effect in renal disease is certainly not met with in eclampsia.

(*b*) By removing toxic substances from the blood and tissues.—The rapid removal of toxic substances from the organism of the patient is a matter of the greatest importance, inasmuch as it is apparent that even if they are not the actual cause of the convulsions, they are always present in large quantities, and their removal is attended by almost immediate improvement. Their removal is effected in the main by promoting the excretory functions of the body. With this object cathartic purgatives are administered. The calomel and jalap powder, as recommended above, if the patient is conscious, is the best purgative. If, however, she is comatose, it is useless to place bulky medicine in her mouth, as it would not be swallowed. In such a case two minims of croton oil made into

a bolus with a little butter, and placed as far back upon the tongue as possible, may reach the stomach. A soap and water, or castor oil and turpentine, enema should also be given if necessary. At the same time the action of the skin must be encouraged. The patient must be kept in blankets, and hot baths administered if possible. If the latter are not possible, a wet-pack or hot-air bath may be tried instead. The amount of urine excreted may be increased by applying hot stupes over the kidneys, while abundance of fluid by the mouth—if the patient is conscious—will also be of use. Diuresis, or at any rate the dilution of the poison, can also be obtained by intravenous or subcutaneous injections of saline solution. For this purpose a solution of the strength of a teaspoonful of common salt to the pint of water is generally used. The same quantity of a mixture of equal parts of bicarbonate of potash and common salt is considered to be better on account of its superior diuretic effect (Jardine). From two to three pints are injected at a time, and repeated as is thought necessary.

In conjunction with saline injections, venesection, to the amount of 17 ounces, has been recommended, with the object of removing some of the toxine-laden blood, which is then replaced by the saline fluid. Whatever may be the value of venesection in attaining this object, it is undoubtedly of use in those cases in which there is marked engorgement of the right heart and pulmonary circulation (Fehling).

(*c*) By emptying the uterus.—Emptying the uterus is said to be a certain method of checking the fits (Dührssen). The author would prefer to modify this statement by saying that if the patient survives the emptying of the uterus, whether by artificial or natural means, she will almost certainly recover. In other words, the prognosis of the case becomes most favourable when the patient is safely delivered. On the other hand, uterine contractions or operative interference will for the time increase the frequency and violence of the fits in the same manner that any other violent movement or emotion will increase them. If the fits can be checked before labour comes on the prognosis of the case is improved. If labour comes on before the fits are checked, the shorter the duration of labour the better the prognosis will be. If the fits continue in spite of all treatment, and labour does not come on of its own accord, the uterus had better be emptied. In such a case the prognosis cannot be made worse, while it may be improved. Following this reasoning, it seems best not to induce labour unless all other means of checking the fits fail. While if labour comes on spontaneously its duration should be shortened as far as possible without adopting too violent measures, that is to say, apply the forceps as soon as the necessary conditions for its application are present.

If it is determined to empty the uterus before the onset of labour, the os is dilated by deep incisions (Dührssen), or, in the case of multiparæ in whom the cervix is long, by combined digital and instrumental dilatation. If the fœtus is dead its extraction may be facilitated by performing craniotomy.

(2) *The Staving off of Complications.*—Complications can to a great extent be avoided by means of intelligent nursing, and by paying the greatest attention to details. While the patient is in a fit she must not be allowed to bite her tongue or otherwise hurt herself. Biting of the tongue is a common accident. Its occurrence can be prevented by the use of a gag of some form placed between the teeth during the fit. A very serviceable gag can be rapidly made by wrapping a towel or other piece of cloth round the handle-end of a spoon. All feeding by the mouth must be stopped while the patient is unconscious. If it is necessary to administer nourishment while she is in this condition, nutrient enemata must be given. The position of the patient must be such that all fluid which tends to collect in the mouth will trickle out at the side of it, instead of running down into the lungs, *i.e.* she must lie upon her side and not upon her back. If the heart becomes weak and rapid, digitalin and strychnine may be administered hypodermically.

Prognosis.—The prognosis for both mother and child in eclampsia is bad, especially for the latter. For the mother the prognosis varies according to the time at which the fits start. It is worse when they commence during pregnancy or labour, it is best when they commence during the puerperium. The greater the number of fits the worse the prognosis. As a rule the occurrence of ten fits constitutes a very severe case. If the child dies, the maternal prognosis is improved (Winckel). The amount of urine passed and the quantity of albumin in it, the presence or absence of marked constipation, the temperature, and the condition of the heart and lungs, are also important guides.

Eclampsia, New Theories of Origin and Methods of Treatment.

Since the preceding article (by Dr. Jellett, of Dublin) was published in 1900, no complication of pregnancy, labour, and the puerperium has been the subject of more extended investigation: new theories of pathogenesis have been advanced, and new methods of treatment founded thereupon have been tried. It cannot, however, be claimed that the cause of this grave condition is yet clear or its treatment satisfactory; it still remains to some extent the opprobrium of the obstetrician. It may be well, however, to indicate briefly these new theories of pathogenesis and methods of treatment, for in some of them may lie concealed

the long-looked-for explanation and cure of this disastrous malady or group of maladies.

Theory of Thyroid Inadequacy. — This theory had its origin in Lange's observations published in 1899 (*Ztschr. f. Geburtsh. u. Gynäk.* xl. 34, 1899). Beginning with the fact of the enlargement of the thyroid gland in pregnancy, Lange noted that in twenty out of twenty-two cases in which this normal hypertrophy was absent there was albuminuria; he noted that thyroidin given to pregnant women in whom thyroid hypertrophy had occurred caused diminution in the size of the gland; diminution in size also followed the administration of thyroidin in a case in which there was pathological enlargement of the gland; and it was found that iodothyrin given to patients suffering from the nephritis of pregnancy was the cause of diuresis although the albuminuria was not much lessened. These observations led Oliphant Nicholson, in October 1900, to test the effect of thyroid extract in eclampsia (*Trans. Edin. Obstet. Soc.* xxvi. 188 *et seq.* 1900-1901), in one case under the care of Dr. Elmslie Henderson, and in another in the Edinburgh Maternity Hospital under Dr. Ballantyne; later, Nicholson reported four additional instances of the use of thyroid extract in eclampsia and albuminuria (*Trans. Edin. Obstet. Soc.* xxvii. 160, 1901-1902). This theory proceeds upon the assumptions that there is always a toxæmia in pregnancy (the result of fœtal metabolism), that this state is more marked towards the close of gestation (causing increased blood tension and cardiac hypertrophy), that errors of diet, etc. may at any time so increase the toxic condition as to cause convulsions, and that although the toxæmia is constant the toxic agent causing the convulsions may differ, and may in some instances be produced by thyroid inadequacy. So long as the kidneys are able to eliminate the toxines no grave symptoms will result, but any checking (especially a sudden one) of the renal functions will produce evil consequences. Dr. Nicholson discussed the pre-eclamptic symptoms and signs (œdema, albuminuria, diminution in the secretion of urine, high arterial tension, headache, eye conditions, diarrhœa, and muscular twitchings), and maintained that they were all explicable on the theory of thyroid inadequacy. He thought, also, that through defect of the thyroid secretion the action of the suprarenal glands might be permitted to become excessive, leading to constriction of arterioles (especially those of the kidney) and increased blood pressure, that the formation of urea ("the most powerful diuretic substance with which we are acquainted") would be diminished and the quantity of urine be lessened, and that the liver, being unable to cope with the untransformed proteids sent to it, would also become inadequate. These factors would combine to produce eclampsia.

For the treatment of eclampsia and the pre-eclampsic state, Nicholson, therefore, recommended and practised the use of thyroid extract in combination with morphia; the morphia was employed for its immediate effect in checking the convulsions, and was given in a dose of half a grain (repeated once or even twice) hypodermically, and the thyroid could be administered before the convulsions or between them (if the patient was able to swallow). The hypodermic injection of the liquor thyroidei might be tried. The dose of thyroid extract ought to be a large one, and the drug may be given till symptoms of thyroidism appear. The effects of this treatment are to relax arterial spasm, to temporarily arrest general metabolism, and so give the thyroid gland time to recover its functions, to supply artificial iodothyrin to counteract the symptoms due to its deficiency, and to supply iodine for the elaboration of iodothyrin in the thyroid gland itself.

The occasional good effects of iodide of potash in the treatment of eclampsia were ascribed by Nicholson to the fact that iodine is a constituent of the thyroid secretion. In a later paper (*Trans. Edin. Obstet. Soc.* xxvii. 160, 1902) Dr. Nicholson brought forward further evidence to show the beneficial effects of thyroid extract in eclampsia, and stated again his belief that some cases of this malady were due to inadequacy of the thyroid and parathyroid glands. In still more recent articles (*Trans. Edin. Obstet. Soc.* xxviii. 84, 1903; xxix. 50, 1904; xxxi. 225, 1906) the subject was further elaborated, and the doses of the thyroid extract increased till in one case of eclampsia Nicholson gave 85 grains in twenty-three hours, followed by 20 grains per diem for three days, and 10 grains per diem for seven days longer.

In connection with this subject Fothergill (*Trans. Edin. Obstet. Soc.* xxix. 41, 1904) has reported a case of maternal eclampsia in which the fœtus, at birth, was found to have hypertrophy of the thyroid gland; the patient was under the influence of chlorate of potash during her pregnancy, and for ten days or so before her labour she had thyroid extract (10 grains daily). In this case the question may be hazarded whether the hypertrophy of the fœtal thyroid had been in any way an attempt by nature to secure amelioration of the inadequacy of the maternal thyroid secretion? Other contributions to the subject have been made by Linn (*Trans. Edin. Obstet. Soc.* xxviii. 78, 1903), by Buist (*Ibid.* xxix. 157, 1904), by Fruhinsholz and Jeandelize (*Presse méd.* Oct. 25, 1902), by Sturmer (*Journ. Obst. Gyn. Brit. Empire*, v. 531, 1904), Macnab (*Ibid.* vi. 386, 1904), Baldowsky (*Vratch*, xi. 1904), Gomot (*Ann. méd.-chir. du centre*, Jan. 15, 1905), and Vassale (*Riv. crit. di clin. med.* March 4, 1905).

Before passing from this theory and its therapeutic application, I may state that while there

is some difference of opinion as to the part played by the thyroid gland and the parathyroids in the production of the convulsions, this does not materially affect the treatment of eclampsia by thyroid extract, for it would seem that thyroid medication remedies troubles due to parathyroid inadequacy.

Theory of Placental or Chorionic Origin.—Much has been said of late in favour of the theory that the toxines that cause eclampsia have their origin in the placenta, but the evidence is not conclusive, for cases of eclampsia occur in the puerperium when there is no longer any placenta in which toxines can be formed, or from which pieces of syncytium can be detached. Leepman (*Münch. med. Wochenschr.* lii. 2484, 1905), apparently, has no doubt regarding the toxic character of a solution made from the placentas of eclamptic patients; he believes that the toxine, which is very labile, is fixed in the protoplasm of the cells of the placenta, and that it is absent from the normal placenta; he is of opinion, also, that the fewer the fits which occur the greater is the amount of toxine in the placenta, while the more numerous are the fits the less toxine is found in the placenta. He thinks that the toxine has a special affinity for the cells of the brain which fix it, neutralise it, and are paralysed by it; that it acts deleteriously on the renal and hepatic parenchyma, and that the lesions in these organs are, therefore, secondary to the toxæmia, and that the epithelium of the chorionic portion of the placenta appears to play an important part in forming the toxine. Leepman surmounts the difficulty of the puerperal cases by supposing that the liver may temporarily store up the toxine during pregnancy and labour to liberate it later. His reason for not accepting the theory that the fœtus is the source of the toxine is the fact that the child itself is so seldom injured by it.

The deportation of villi or of parts of villi from the placenta into the mother's blood is a closely allied theory to that stated above, but it is very largely theoretical. Labhardt (*Zeitschr. f. Geburtsh. u. Gynäk.* liv. 264, 1905) discusses this view and the modifications of it, and groups them into three classes—(1) Schmorl thought that placental cells passed into the maternal circulation; Ehrlich had shown that the entrance of foreign cells into an organism caused chemical changes; and Veit believed that the placental cells produced a poison in the mother which caused eclampsia. Since only a few cells usually passed over very little poison was produced, and so eclampsia rarely followed. (2) Ascoti expanded this view, and stated that the foreign cells brought about the formation of new substances which dissolved them and neutralised their poisonous effects; these new substances were lysins (syncytiolysins); if they are formed *in excess* they cause eclampsia. (3) Weichardt, again, thought that by the solution of the placental cells a new poison was formed (syncytiotoxin), which is usually neutralised by an antitoxine, and that if the antitoxine fail to be formed in sufficient amount the syncytiotoxine causes the eclamptic seizures. Labhardt criticises these views, pointing out that deportation of villi is frequent, while eclampsia is rare, that the placental cells are hardly "foreign" to the maternal organism, that eclampsia is very rare in animals which possess placentas, and that the frequency of eclampsia in primiparæ, in cases of flat pelvis and hydramnios, and in certain places and at certain times, is not accounted for by this theory. Its non-occurrence in cases of tubal abortion is also a mystery.

It must be admitted that the proof of the placental theory of origin of eclampsia is defective. In the meantime, however, in Bumm's clinique, obstetric practice proceeds along the lines which are obviously indicated by such a theory if it be correct, viz. the immediate emptying of the uterus. Up to 1895 the treatment there was *purely* symptomatic by means of narcotics, etc., and the mortality was 30 per cent; from 1895 to 1900 narcotics were still used, with hot packs, venesection, and the use of saline infusions, and the mortality was 30 per cent; since 1901 immediate delivery (by hysterotomia anterior if the os were undilated, by forceps or version or perforation in other cases) has been practised, narcotics and chloroform being sparingly used, and venesection and the use of saline infusions and hot packs being employed as subsidiary agents. Out of 104 cases so dealt with the mortality has been 2·8 per cent (three deaths having occurred); but it is noteworthy that some fatal cases were excluded from the lists. Elsewhere than in Berlin and Halle the treatment of eclampsia by rapid delivery seems to be in favour. De Lee (*Internat. Clinics*, s. xvi. vol. i. p. 145, 1906), for instance, believes in emptying the uterus as rapidly as is consistent with the safety of the mother. Zweifel also (*München. med. Wchnschr.* liii. 297, 1906), believing as he does that the cause of eclampsia is fœtal (deficient oxygenation), empties the uterus as soon as he can. Dienst (*Zentrlbl. f. Gynäk.* xxix. 353, 1905) regards the causal factor in eclampsia as the mixing of the fœtal and the maternal blood, for he found the placenta in such cases to be more permeable, and he thinks that blood from the fœtus acts upon that of the mother like the blood of a distinct species. In support of his views, Dienst discovered that blood obtained by venesection from two cases of albuminuria, when tested upon normal blood, caused agglutination and hæmolysis; after delivery, the maternal blood was tested on that of the infants for agglutination, with a positive result. It must, therefore, from these and many other observations, be admitted that the theory of the fœtal or placental origin of the toxæmia of eclampsia, although not established,

is far from being discredited, and that there are, in consequence, reasons still in force in support of the rapid termination of pregnancies in which eclampsia develops.

New Methods of Treatment.—It cannot be said that any of the new methods of treatment which have been introduced within the last few years have been shown markedly to reduce the mortality from eclampsia. The introduction of *Bossi's metallic dilator* of the cervix gave a rapid and in proper cases (*i.e.* cases in which the cervix was taken up) a safe method of dilating in order quickly to empty the uterus, but it does not appear that the results following its use are such as to justify its employment in the cases in which the cervix is not taken up, for in them the risk of severe lacerations is considerable.

In order to avoid the risks associated with rapid dilatation, the operation of *vaginal section* (often called, somewhat misleadingly, vaginal Cæsarean section) has been practised. With the patient in the lithotomy position the operator exposes the cervix (with a speculum) and draws it down to the vulva (with volsellæ). If the patient be a primipara, with a narrow vulva and vagina, it may be well to do an episiotomy at once to give room. An incision is then made through the mucous membrane of the anterior lip of the cervix and the anterior vaginal wall of an inverted T (⊥) shape. The bladder is now separated from the cervix by the fingers or the handle of the knife until the peritoneum of the utero-vesical pouch comes into view; the latter is not incised. The cervix is next split in the middle line with scissors, pulled farther down with the volsellæ, and the incision (4 inches long) is carried farther up into the lower uterine segment. If the operation be near the full term it will be well to incise the posterior as well as the anterior lip of the cervix. Bleeding is controlled during the making of the incisions by downward traction upon the cervix. The delivery of the child is now rapidly accomplished, the placenta removed, and the uterus massaged and (perhaps) packed with gauze. The incisions in the cervix are closed with sutures (continuous or interrupted catgut) placed near together; the posterior incision (when present) is closed first. Care must be taken that the sutures do not catch in the gauze packing in the uterus. The vaginal incision is closed by continuous catgut suture, and the perineum is repaired (if episiotomy have been performed).

In order to check the convulsions and give time for medicinal means to have effect, *lumbar puncture* has been employed. The theory of its mode of action is very simple: it is taken for granted that the convulsions are due to increased cerebro-spinal tension; therefore, if some of the cerebro-spinal fluid be removed by puncture of the arachnoid space the tension will be diminished, and the fits will for the time cease; this interval of freedom from fits may be utilised for the application of other forms of treatment which will effectually and permanently remove the cause which has led to the high tension. Dr. Helme of Manchester used this treatment in December 1903 (*Brit. Med. Journ.* i. for 1904, p. 1131), in a typically severe case of eclampsia in which saline injections, thyroid extract, and chloral had previously been given, and the patient recovered. It was noted that the fluid escaped from the canal as if under considerable pressure. Three cases treated in the same way by B. Krönig also recovered (*Zentrlbl. f. Gynäk.* xxviii. 1153, 1904); but out of sixteen cases dealt with by Max Henkel four terminated fatally (*Zentrlbl. f. Gynäk.* xxviii. 1329, 1904). I reported a case in which I used lumbar puncture in October 1904 (*Trans. Edin. Obstet. Soc.* xxx. 132, 1904-5); it was a grave case, and proved fatal. I noted that the fluid did not escape freely from the needle, oozing out drop by drop. I have since used it in another grave case, which also terminated fatally, and in it, also, the cerebro-spinal fluid escaped drop by drop. It does not appear, therefore, as if treatment by lumbar puncture could claim any superiority over other methods. A recent communication on the subject is that by Proud (*Brit. Med. Journ.* i. for 1906, p. 678); in two cases recovery followed the puncture. It was also carried out in a remarkable case reported by Jardine in which 200 fits occurred, and the patient recovered (*Journ. Obst. Gynæc. Brit. Empire*, x. 38, 1906).

Another surgical method of dealing with eclampsia, *renal decapsulation*, was introduced by Edebohls in 1903 (*Amer. Journ. Obstet.* xlvii. 783, 1903; l. 260, 1904). It was recommended also by Sippel (*Zentrlbl. f. Gynäk.* xxviii. 479, 1904). Its object is to diminish intra-renal tension. Chambrelent and Pousson perform incision of the kidney substance (nephrotomy) as well as splitting of the capsule (*Gaz. hebd. d. sc. méd de Bordeaux*, xxvii. 173, 1906); and Pinard thinks it should be limited to cases in which there is complete anuria (*Bull. acad. de méd.* 3 s. lv. 489, 1906).

Eclampsia Nutans.—Salaam convulsions. *See* Head-Shaking.

Eclecticism.—In ancient times there was a class of philosophers who, without attaching themselves to any one school of thought, "selected such doctrines as pleased them in any school" (Gr. ἐκλέγειν, to select); so, in modern use, the word has come to mean a system (*e.g.* of medical practice or of therapeutics) unfettered by the rules or opinions of any special form of theory or practice. An eclectic remedy or drug is one to which popular belief attributes certain curative qualities or powers, although scientific medicine does not or cannot explain its mode of

action or its good effects on any recognised principles of pharmacology or therapeutics.

Eclimia.—Excessive hunger (Gr. ἐκλιμία) or inordinate appetite; bulimia. *See* Appetite (*Increase*).

Ecmnesia.—A variety of amnesia in which occurrences prior to a certain date are remembered perfectly, while those happening for a certain time after that date cannot be recalled.

Economy.—The organisation or the harmonious working of any complex living body, such as an animal or a plant (Gr. οἰκονόμος, one who manages a household).

Écouvillonage.—A gynecological operation in which the interior of the uterus is scrubbed out with an *écouvillon*, or brush (consisting of a wire stem with strong, short quills projecting from it), especially in cases in which it is suspected that puerperal debris is present, and is setting up morbid intra-uterine processes and causing sapræmic symptoms. *See also* Curettage.

Ecphronia.—Insanity (Gr. ἔκφρων, senseless), of a maniacal (*e. mania*) or melancholic kind (*e. melancholia*).

Ecphyadectomy.—Removal of the vermiform appendix (Gr. ἐκφύω, to grow from, and ἐκτέμνω, I cut out); appendectomy. *See* Appendicitis.

Ecphyma.—A blister, pimple, wart, corn, or callosity (Gr. ἐκφύω, I grow from).

Ecplexis.—A state of "stupor or consternation, the patient standing with open eyes and mouth, speechless and motionless" (Hack Tuke); it is derived from ἔκπληξις, panic fear.

Écraseur.—An instrument (Fr. *écraser*, to crush) for the separation (bloodlessly) of parts or tissues by gradual crushing; the apparatus consists of a chain, wire, or cord, which can be slowly tightened in a regular fashion by means of a graduated screw or rack and pinion; the wire may be heated and the écraseur be made to burn the tissues (galvanic écraseur).

Ecstasy.—As the derivation (Gr. ἐξιστάναι, to put out of place) indicates, the word ecstasy literally means the state in which one is beside oneself; it has come to mean specially that morbid condition in which the powers of locomotion and sensation (the lower cerebral and some reflex functions) are, for the time, suspended, while the mind (the upper cerebral functions) is exalted in its action and occupied exclusively with one idea (*e.g.* religious joy or sorrow, or rapturous delight); cataleptic trance. *See also* Catalepsy; Hypnotism; Hysteria; Hysteria in Childhood; Insanity, Nature and Symptoms (*Cataleptic Stupor*); Spasm (*Hysterical*).

Ecstrophy.—Eversion or extroversion, *e.g.* of the bladder. The word is derived from the Gr. ἐκ-, or ἐξ-, out, and στρέφειν, to turn, and, although it is often spelt "exstrophy," yet, according to Murray's *New English Dictionary* (vol. iii. p. 453), it ought, according to the analogy of the Greek derivatives, to be "ecstrophy," as above. It is usually applied now almost solely to the congenital malformation of the bladder (defective formation of the anterior vesical wall), in which that organ appears to be turned inside out. *See* Bladder (*Malformations, Ectopion vesicæ*); Ectopia; Extroversion; Teratology; etc.

Ectad and **Ectal.**—Both these words are derived from the Gr. ἐκτός, without; the former means toward the exterior, and the latter means belonging to or associated with the exterior (*e.g.* of a cell).

Ectasin.—A substance acting as a vasomotor dilator; *ectasis*, meaning distension or dilatation, occurs in such compound words as *telangiectasis*, and also, occasionally, alone.

Ectental.—An embryological term; in the blastula stage of segmentation the line along which the ectoderm and entoderm join is called the ectental line.

Ecthol.—A proprietary medicine recommended in erysipelas, and said to act as an antiseptic; it is a preparation of *Thuja occidentalis* and *Echinacea angustifolia*.

Ecthyma. *See* Impetigo (*Impetigo Contagiosa of Tilbury Fox*); Skin, Bacteriology of (*Ecthyma*).—This is not the name of any independent cutaneous disease, but is rather that condition in which there are deep-seated pustules, in which thick crusts form, and in which cicatrices or pigmentary marks are left; it is due to pyogenic cocci.

Ecto-.—Ecto- (from the Gr. ἐκτός, without) forms part of many compound words, and signifies *without* or *external*; thus *ectoblast* is the outer layer of a cell, *ectoderm* is the outer layer of the blastodermic vesicle or the epiblast, *ectomere* is one of the cells of the ectoderm, and *ectozoon* is an ecto-parasite (one affecting the outer aspect of the body of the host).

Ectocardia.—Displacement of the heart, or *ectopia cordis*. The heart may be displaced to one side of the thorax, or down into the abdomen (through an opening due to defective development of the diaphragm), or outside the chest (due to defective development of the chest wall, such as fissure of the sternum or of the side wall of the chest).

Ectogenous.—Capable of developing outside a host, but usually parasitic, *e.g.* certain micro-organisms.

Ectopagus.—A form of double monster, in which the twins are united by the lateral aspect of the thoraces (*Gr.* ἐκτός, without, and πάγιος, fixed); there may be two arms (dibrachius), or there may be three (tribrachius), of which one is median and may show signs of duplicity. *See* Teratology.

Ectopia.—Displacement (*Gr.* ἔκτοπος, distant, out of the way), *e.g. ectopia cordis* (displacement of the heart, ectocardia), *ectopia lentis* (dislocation of the crystalline lens), *ectopia testis* (the presence of the testicle in the inguinal canal, in the perineum, or in the abdominal cavity), and *ectopia vesicæ* (defective development or eversion of the urinary bladder). *See* Bladder, Injuries and Diseases (*Malformations, Ectopion*); Chest, Deformities (*Congenital Deformities*); Heart, Congenital Malformations (*Ectopia Cordis*); Labour, Faults in the Passenger (*Child, Ectopia Viscerum*); Lens, Crystalline (*Displacements*); Scrotum and Testicle, Diseases (*Ectopia Testis*); Teratology (*Malformations of the Thorax and Abdomen, and Contents*).

Ectopic Gestation.

See also Abortion (*Diagnosis*); Menstruation and its Disorders (*Menorrhagia and Metrorrhagia*); Ovaries, Diseases (*Diagnosis*); Pelvis, Hæmatocele and Hæmatoma (*Diagnosis*); Pregnancy, Hæmorrhage (*Ectopic Gestation*).

There is probably no pathological condition of which more has been learned within recent years than extra-uterine pregnancy. To the late Mr. Lawson Tait is due the honour of having, twenty years ago, commenced a scientific investigation of the condition, and of having devised satisfactory methods of dealing with it. But the abnormality was recognised even centuries ago, and the earliest writers mention this form of fœtation as a curiosity, although they offer no explanation as to its cause. The remarkable book by Parry, published in 1876, in which he recorded 500 cases, was, however, that which gave the first impetus to the advance of our knowledge of ectopic gestation. Several cases had been operated on before this time, but it was not till Lawson Tait operated in 1883 that a definite attempt was made to check the hæmorrhage from a recently ruptured extra-uterine pregnancy, and so save the life of the mother.

Under ordinary circumstances the normal site for the growth and development of the impregnated human ovum is within the cavity of the uterus. But it not infrequently happens that the ovum develops outside the uterus, and when this occurs, no matter whether it be found in the Fallopian tube, in the uterine wall, or elsewhere, the condition is known as "Extra-Uterine Pregnancy," or better as "Ectopic Gestation." (French, *Grossesse extra-uterine;* German, *Extra uterinschwangerschaft.*)

For purposes of description and discussion it is convenient to group these ectopic gestations under three distinct heads: 1. Abdominal; 2. Ovarian; 3. Tubal.

I. *Abdominal.*—The possibility of the occurrence of a primary gestation sac free in the peritoneal cavity has been denied by many authors. It is conceivable that a spermatozoon, after worming its way up through the uterine cavity and through the whole length of the Fallopian tube, might at last emerge from the fimbriated end, and meeting an ovum which had remained free in the peritoneum, might fuse with it. This is certainly conceivable, but its rarity is such as to practically put it out of court at once as unworthy of consideration. It is certainly of no practical importance, and is to be regarded purely as a curiosity. Very few observations as to the possibility of its occurrence have been made, but the case of Schlectendahl seems to indicate that it might occur.

II. *Ovarian.*—This form of ectopic gestation is also rare, and its possibility is denied by many; however, it is difficult to disprove the cases recorded by Leopold, Patenko, and Martin.

Leopold removed a pelvic tumour of twenty-five years' standing, which proved to be an ovary containing a lithopœdion.

Patenko removed an ovary the size of a hen's egg; it contained a cyst in which were found fœtal bones.

Martin of Berlin has also recorded two similar cases, and advances the suggestion that the spermatozoon finds its way to the abdominal

ostium of the tube into one of the recently ruptured follicles on the surface of the ovary, and that there it coalesces with the ovum.

But for all practical purposes tubal is the *only primary* form, and falls now to be considered in detail.

III. *Tubal.*—(*a*) Causes.—The conditions which lead to the production of this abnormality are various; and first one meets the unsettled difficulty of determining where impregnation normally takes place. The older writers believed, and some modern authorities agree with them, that impregnation usually took place in some part of the Fallopian tube, whereas recent writers allude to impregnation as more frequently occurring in the uterus itself. Indeed, Lawson Tait goes the length of stating that it never normally occurs elsewhere. As to the actual causes which bring about the development of a fertilised ovum in the Fallopian tube, prominence distinctly has been given to inflammatory changes in the tube itself, destruction of the cilia—desquamative salpingitis,—and that there is truth in this is proved by the numerous cases in which tubal pregnancy occurs in patients who have suffered from tubal disease. Bland-Sutton, however, and Taylor hold that this condition as a cause does not exist. The former, indeed, goes the length of saying that a healthy Fallopian tube is more likely to become gravid than one that has been diseased. Tait gives a very interesting account of his theory, showing that as the action of the cilia in the tube is from the ovary down to the uterus, the onward movement of the ovum is facilitated, while the upward movement of the spermatozoon is materially interfered with. In a case, however, of desquamative salpingitis, where the cilia are absent, the spermatozoon easily enters the tube and reaches the ovum, which is fertilised and develops there. It has, however, recently been proved that the movement of the cilia in the uterus is from above downwards, and not from below upwards, as has been previously supposed, so that Tait's ingeniously simple theory cannot now be accepted.

Congenital deformity of the tubes, showing itself in excessive tortuosity, or the presence of a diverticulum, no doubt acts occasionally in producing the condition; and, further, a simple inflammatory swelling of the mucosa is no doubt a cause of obstruction to the onward passage of the ovum, especially in the middle and outer segments of the tube.

Cases have also been recorded, but they are, of course, rare, where a polypus or a myoma has acted as a valve, and so blocked the inner end of the tube.

In addition to this, it is true that abnormal development of the tube, causing decreased muscular power and peristaltic movement, may delay the passage of the ovum.

(*b*) Varieties.—The Fallopian tube, considered anatomically, naturally divides itself into three distinct parts:—

1. Interstitial,—that part of the tube which lies in the thickness of the uterine wall.

2. Infundibular,—the expended part of the tube close to the fimbriated extremity.

3. Ampullar,—the main portion of the tube which stretches between the two first-mentioned parts.

These three parts of the tube give their names to the three forms of tubal pregnancy, according as the ovum settles in one or other of them. We must now discuss each of these forms in turn.

1. *Interstitial tubal pregnancy* or *tubo-uterine* is really not ectopic pregnancy at all. Taylor very correctly divides tubo-uterine pregnancy into two varieties:—

(1) Primary interstitial pregnancy.

(2) Secondary, caused by invasion of the uterine wall by a growing tubal pregnancy.

Many cases of so-called interstitial pregnancy have been recorded, but they are generally by older authors, and recent specimens of true tubo-uterine pregnancy are extremely rare, and probably these early recorded cases have been really cornual pregnancies.

In this variety the environments of the gestation sac differ from those in ampullar and infundibular pregnancy. The muscular fibres hypertrophy, and though rupture is almost inevitable, it does not occur so early as in the two other forms. Occasionally the fœtus escapes into the uterus, and is either expelled at once or goes on to full time, and is then born in the natural way, but definite proof of these possibilities has never been obtained.

Rupture much more frequently occurs into the abdominal cavity with profuse hæmorrhage and a fatal termination in a very short time.

It is unquestionably the most dangerous of all the forms of ectopic gestation, fatal rupture generally occurring about the fifth month.

It has been observed that in this variety a remarkable asymmetry of the uterus is found, due to the invasion of the fundus uteri by the growing ovum, and on this account the condition may have been mistaken for a cornual pregnancy.

2. *Infundibular Tubal Pregnancy.*—The ovum may become implanted in the outer third of the Fallopian tube in the infundibulum, and in such cases various possibilities present themselves.

The abdominal ostium is not so liable to become hermetically sealed in this as in the other varieties, so that free hæmorrhage may occur from the seat of the pregnancy into the abdominal cavity. Occasionally the ovum is so lightly attached within the fimbriated extremity that it is extruded entire into the peritoneal cavity accompanied by hæmorrhage, but without any actual rupture of the tubal walls. This

condition is known as *tubal abortion.* The ostium is found enlarged and patulous, and the abortion lies in the free blood in the abdominal cavity. On the other hand, when the ovum is situated *near* the fimbriated extremity its life-history may be the same as that of an ampullar pregnancy (to be considered later), resulting in development within the tube, rupture, intra-peritoneal or extra-peritoneal, or the formation of a *tubal mole.*

3. *Ampullar Tubal Pregnancy.*—This variety, in which the ovum is implanted in the middle portion of the tube, is the commonest of all, and the changes which take place in and around it fall now to be discussed in detail. These changes are, to a greater or less extent, common to all varieties of tubal gestation.

During the first few weeks the portion of the tube which contains the impregnated ovum becomes very much swollen and extremely vascular. The muscular tissue is also hypertrophied, but after the sixth week, and indeed, in some cases from the very commencement of pregnancy, a process of thinning sets in. The mucous membrane becomes stretched, and its plicæ disappear, while the muscular tissue becomes thinned and to a certain extent replaced by fibrous tissue. As the pregnancy advances the wall of the tube appears as a mere transparent membrane composed of the attenuated layer of muscular and fibrous tissue and of peritoneum.

At the same time important changes are taking place at the abdominal ostium. It shares in the congestion and swelling of the parts; the fimbriæ become turgid; the peritoneum surrounding the ostium becomes more and more swollen, until it projects as a ring beyond the fimbriæ, and finally about the eighth week contracts and hermetically closes the ostium. The condition of the ostium affects the subsequent history of the pregnancy, for, although a gravid tube with a patent abdominal ostium may rupture, there is always a possibility of "tubal abortion." If the ostium is occluded rupture is inevitable. The changes at the uterine end of the tube have not been so thoroughly investigated, but in a few instances the lumen has been found patent.

In all probability, until there is some attempt at a differentiation of the placental site, the ovum is freely movable. But afterwards, if the ovum goes on growing and a placental site is determined, the relations between the ovum and tube become very close and intimate. The ovum is soon embedded in the thickened mucous membrane, which gradually loses its characteristic plicæ, but probably no genuine decidual tissue is formed as was originally suggested by Webster. Where the ovum settles the chorionic villi dip into the mucosa as they do in ordinary pregnancy into the decidual serotina. The fœtal membranes develop exactly as in the case of intra-uterine pregnancy. In a normal pregnancy the placenta is composed almost equally of decidua serotina and chorion frondosum, but in tubal gestation "the mucous membrane plays a very insignificant part, the placenta being mainly if not entirely derived from the fœtal tissues" (Bland-Sutton).

Until primary rupture takes place the placenta is developed entirely in relation to the mucous membrane lining the tube, but after this event, if the pregnancy still goes on, important modifications take place.

The effects of rupture on the placenta have been especially treated by Hart, who points out that if the embryo be situated above the placenta the latter becomes depressed between the layers of the broad ligament until it reaches the pelvic floor. If, on the other hand, the embryo is below the placenta, the latter is pushed up until it lies high in the abdomen.

From a study of extra-uterine placenta Hart points out that in tubal gestation the villi are embedded in decidual cells; there is no inter-villous sinus system, but large sinuses are found in the muscular wall. When the placenta is displaced from extra-peritoneal rupture it becomes damaged, and is reduced to a mass of compressed villi, and its function is extensively impaired. The placenta sustains the least damage when the embryo is situated above it, because then there is less displacement, and further, when the placenta grows up into the abdomen there is much greater risk of intra-peritoneal rupture and of fatal hæmorrhage.

With regard to the effect on the fœtus of the changes in the site and development of the placenta, the placenta performs its function inefficiently in any case, but when situated below the embryo, in the mesosalpinx, the nourishment of the child is not so greatly interfered with.

A fœtus, even when delivered by the surgeon at or near full time, rarely lives more than a few days or weeks, and is defective in size, or presents some more or less evident malformation.

During the early weeks of extra-uterine pregnancy changes of hypertrophy and development are also in progress in the uterus itself, similar to those which occur during the early months of a normal pregnancy. The organ increases in size up till the third month of extra-uterine pregnancy, and although no decidual membrane forms within the Fallopian tube around the growing ovum, a thick, soft, spongy decidua develops within the uterine cavity. This decidua is often expelled; it may be entire, when it is a complete cast of the interior of the uterus—the inner surface smooth and the outer surface rough and shaggy—or it may be discharged in fragments. This passage of decidual membrane in whole or in part is a most important sign in diagnosis, as we shall see later.

The time when the membrane is passed varies. It is very seldom that it is retained until full term and passed when false labour sets in. More frequently it is discharged in the early weeks, whether the gestation goes to full term or not. Its passage may be accompanied by hæmorrhage and pain, but the pain may be entirely absent. The author's own experience has not led him to associate the passing of the decidua with any special change in the pregnancy.

Further History of a growing Tubal Pregnancy.—The possible results of a tubal pregnancy are shown in the following table :—

1. The ovum may die early in the tube owing to its foreign surroundings, and as a result may form a hæmatosalpinx and a tubal mole, or may be the cause of an intra-peritoneal hæmatocele.

2. The tube may rupture.

(a) Early rupture—2nd to 6th week. Woman has some shock, but is not seriously ill, and may recover quickly. Fœtus, etc. is absorbed.

(β) Later rupture—8th to 12th week.

(1) Into peritoneal cavity. Woman usually dies in a few hours if not operated upon. If mother survive : (a) Fœtus may live and may go on to full term if placenta remains attached. (b) Fœtus more often dies and becomes absorbed ; lithopœdion, etc.

(2) Between layers of broad ligament. Woman seldom dies : (a) Fœtus may live and grow, becoming subperitoneo-pelvic, and later subperitoneo-abdominal. (b) Fœtus may die and undergo any of the usual changes. (c) Sac may rupture a second time intra-peritoneally, and this is usually fatal to the mother.

1. Tubal Mole. — During the development of a tubal pregnancy the life of the fœtus is in constant danger, and is especially so until the fœtal membranes are well formed. The chorionic villi have but a loose attachment to the tube and are easily separated. Hæmorrhage into the sac or between the membranes may occur and prove fatal to the continuation of the gestation. This when it takes place very early (from the first to the third week) is the most favourable termination possible for the pregnancy, and may give rise to practically no symptoms whatever, and the patient may experience no discomfort. Such an apoplectic ovum appears at first sight as simply a blood-clot, but such bodies are known as "tubal moles," and careful examination reveals their true character. If the pregnancy is further advanced when the hæmorrhage into the ovum and the formation of a mole occurs, the attending danger to the mother is much greater, as the accompanying hæmorrhage into the tube may cause rupture of the tube and the rapid death of the patient. Tubal moles, if recent in origin, are found to contain an embryo. But after a time, especially if the mole has been extruded into the abdominal cavity or into the broad ligament, the fœtal tissue may become completely disintegrated, and it may be impossible to recognise any of these except the chorionic villi, which are most persistent, and may be found long after all evidence of their origin has disappeared. These villi present the same appearances as those of a uterine pregnancy.

If the blighted ovum remains in the tube, though the pregnancy becomes abortive it is by no means harmless. It causes constant irritation of the tissues to which it is attached, and may be the cause of frequent hæmorrhages into the abdominal cavity and into the lumen of the tube, while inflammatory changes are set up in all the organs, which become matted together, and along with blood-clot and inflammatory deposit form a complete mass.

Intra-peritoneal Hæmatocele.—No doubt the commonest cause of intra-peritoneal hæmorrhage is a tubal pregnancy ; it may be the result of rupture or of a bleeding from the fimbriated extremity of the Fallopian tube. The latter cause is the more frequent, and is caused by the presence of a mole in the tube. In these cases of hæmorrhage from the ostium it is generally moderate in amount, and a well-defined hæmatocele is formed. In the case of rupture the hæmorrhage is so copious and so diffuse that unless the patient is operated on she dies before there is time for the formation of a defined blood tumour. Taylor gives the following statistics :—Out of 21 cases of intra-peritoneal hæmatocele due to tubal pregnancy, 14 were due to hæmorrhage from an unruptured tube, while 7 were associated with rupture. Cullingworth states :—"Of 25 cases of pelvic hæmatocele in which an opportunity occurred of verifying by actual inspection the source of the bleeding, 23 were instances of hæmorrhage from the open abdominal ostium of a pregnant Fallopian tube, and only 1 was due to rupture."

Blood effused into the abdominal cavity comes to form a definite tumour by local peritonitis being set up around it and binding the pelvic and abdominal viscera to each other and to the blood-clot. The outer layer of the blood-clot solidifies and forms a capsule which may in turn rupture, allowing a fresh intra-peritoneal hæmorrhage to occur. On the other hand, the blood within the sac may be absorbed and only the capsule remains adherent to the Fallopian tube.

2. Rupture. — (a) *Early Rupture.* — It has been stated that one result of a tubal pregnancy is the formation of a "tubal mole," and the possible results of that condition have been described. But very frequently an accident occurs at a still earlier period than that at which a mole is formed, which terminates the pregnancy. This is what is known as *early rupture* of the tube. It occurs from the second to the sixth week, and is quite distinct from what is known as *later rupture*, which takes

place from the eighth to the twelfth week, in that it is much less serious in its results and the patient usually recovers.

Early rupture may occur at any part of the tube, except at the fimbriated extremity, but the most common site is at the isthmus, *i.e.* just where the tube becomes free from the uterine wall. This early rupture has been shown to occur in cases where the tube is atrophic, and specimens removed have demonstrated the fact that in these cases the usual primary thickening of the tubal wall has not taken place. These facts obviously account for the occurrence of this early rupture. Such cases are characterised by the suddenness with which the symptoms come on. There is no previous warning, but the patient becomes rapidly collapsed, and shows all the signs of internal hæmorrhage. The patient may, however, speedily recover without any active interference.

(*b*) *Later Rupture.*—If the ovum escapes the possibilities of "early rupture" or the formation of a "tubal mole" it is certain that "later rupture" will occur. This takes place, as we have already stated, between the 8th and 12th weeks. This rupture is nearly always associated with hæmorrhage more or less severe, the amount depending on the relation of the placenta to the exact seat of rupture. When the rupture involves the placental site, or causes separation of the placenta, the hæmorrhage is severe, though not necessarily immediately fatal. If, on the other hand, the attenuated tubal wall alone is torn the bleeding may be slight or even absent altogether.

Owing to the anatomical relations of the tube there are practically only two directions in which the blood effused from a rupture may be extravasated, and these are :—

1. Into the peritoneal cavity, and
2. Between the layers of the broad ligament.

In the former case the hæmorrhage may be so severe and so rapid as to prove immediately fatal to the mother, but in some cases, when the bleeding is more gradual, a hæmatocele, to a certain extent encapsulated by a plastic peritonitis being set up around it, is formed. If, however, there is further bleeding the capsule of this hæmatocele may in turn give way, and the abdominal cavity may rapidly be filled with blood. This form of hæmatocele is quite distinct from that occurring from a "tubal mole," in which the hæmorrhage from the abdominal ostium is so gradual that a capsule is formed consisting not only of peritonitic adhesion, but also of organised blood-clot.

In the second case, when the hæmorrhage occurs between the layers of the broad ligament, it is not nearly so profuse, being now extravasated into the interstices of a cellular tissue. This very soon becomes firm owing to the coagulation of the effused blood, and constitutes an effectual barrier to further hæmorrhage.

The Fœtus after Rupture.—No matter whether the matter be intra- or extra-peritoneal, the fœtus in most cases follows the course of the hæmorrhage and escapes through the rent, and the pregnancy now becomes either tubo-abdominal or tubo-ligamentary. It has been a matter of dispute under what conditions the fœtus may survive after rupture, some authorities holding that for the continuance of its life the amnion at least must remain intact. Others, however, believe that even this is unnecessary, and aver that it may develop even to full term while lying free in the abdominal cavity.

1. When the pregnancy becomes tubo-abdominal, the fœtus having escaped into the abdominal cavity, it may, if it survive the rupture of the tube, go on to full term. At or about that time, if not delivered by abdominal section, it succumbs and undergoes various changes.

On the other hand, and more commonly, the fœtus dies at the time of rupture, and if the mother survive those changes merely occur at an earlier date.

For example, a dead embryo up to the second month lying free in the abdominal cavity may be completely absorbed. If it is beyond the second month it may—

(*a*) Decompose and form an abscess which may burst externally or into any neighbouring viscus.
(*b*) Become mummified.
(*c*) Become calcified and form a lithopœdion.
(*d*) Be converted into adipocere and form a lipopœdion.
(*e*) Disappear partially, only the bones remaining, forming an osteopœdion.

2. When the pregnancy becomes tubo-ligamentary the fœtus may die, owing to the pressure exerted on the placental circulation by the blood-clot, and in this case it remains between the layers of the broad ligament, and may undergo any of the above changes.

But very often the hæmorrhage after rupture is but slight, and the fœtus continues to develop in its new situation. It gradually increases in size, but is largely confined to the pelvis by the limits of the broad ligament and by the placenta above it. In this position it is described as subperitoneo-pelvic.

The pregnancy may continue in these relations, but it is the exception that these cases should go on to full term. In the great majority of cases, either the fœtus gradually separates the peritoneum from the abdominal wall, and grows up into the abdomen extra-peritoneally, when it is described as subperitoneo-abdominal, or what is known as *secondary rupture* occurs, that is to say, the broad ligament gives way and the fœtus escapes into the abdominal cavity, exactly as in the case of primary tubo-abdominal rupture. Cases of secondary rupture are always extremely grave. In the case of a primary tubo-abdominal

rupture, ligature of the broad ligament and removal of the tube arrest the hæmorrhage, but in cases of secondary rupture separate vessels must be secured, the placenta is much more difficult to deal with, and one has to trust largely to pressure and drainage.

General Symptoms and Diagnosis of Tubal Gestation

The difficulties which attend the accurate diagnosis of early extra-uterine pregnancy are numerous, and are for the most part well recognised, and indeed, from the large proportion of cases which terminate fatally if left undiagnosed and untreated, it is clear that the value of an early and accurate diagnosis can scarcely be exaggerated. It formerly was commonly stated that diagnosis before rupture was quite impossible, but this is nowadays, with our increased knowledge of the subject, quite inaccurate. Indeed, the proportion of cases in which the condition is diagnosed is an increasingly large one, and Taylor records that out of his 42 cases the exact nature of the lesion was recognised in 32 before operation. Extra-uterine gestation may occur at any age during the period of a woman's fertile life. As a rule it will be found that the patient has been in fairly good health before the pregnancy occurred. Even though there be a history, as there often is, of previous tubal disease, this has evidently been in a quiescent state for some time. In about one-half of the recorded cases it has been noted that usually several years have elapsed since the last normal pregnancy took place, and this point always excites a certain amount of suspicion if any other symptom of extra-uterine gestation is present.

The general constitutional symptoms of normal pregnancy may or may not be present. It not infrequently happens that the subjective symptoms are entirely absent, and the patient may be quite unaware of her condition. More commonly, however, one finds the usual mammary changes with the formation of the areola, but Montgomery's tubercles are seldom well developed. Gastric disturbance may be evident, the patient frequently complains of pelvic pain, usually confined to one side, and there is practically always a history of some derangement of menstruation.

As a rule one finds that the patient has missed one or two periods, and then has suffered from irregular hæmorrhages. Cullingworth has drawn attention to the character of these hæmorrhages. He says, "The blood is almost invariably dark in colour, moderate in amount, thickish in its consistence, and steady in its rate of flow. Gushes of bright red blood occur occasionally, but are quite exceptional." In the writer's experience, however, floodings are not so very uncommon.

Accompanying these hæmorrhages shreds of membrane are frequently passed, but in other cases the uterine decidua is passed entire. The presence of these shreds, or of the whole decidua, forms a most important sign, but is one which unfortunately cannot always be obtained. If the membrane be examined, it is important not to mistake it for that passed in the rare condition known as "membranous dysmenorrhœa," in which the membrane is usually expelled in small pieces and rarely as an entire cast. The membrane from the uterus in cases of extra-uterine gestation is much thicker, and is generally shed either in large pieces or as a complete cast of the interior of the uterus. The history of the case, too, would be of great value in differentiating between the two conditions.

(*a*) *Diagnosis before Rupture.*—The character of the pelvic pain varies. It may be dull and aching, or it may be, and is especially just before rupture takes place, sharp and lancinating.

The external genitals and the vagina may present the same appearances as in normal pregnancy, and on bimanual examination, the uterus enlarged, soft, and globular, is displaced to one or other side of the pelvis by a swelling which can be felt behind and to one side of the uterus. This swelling is tense, elastic, and fluctuating, and through the vaginal roof—a most valuable sign—the presence of pulsating vessels may be detected on the affected side. The swelling during the first few weeks may be movable and insensitive, but later it becomes fixed and tender. Another and an important point in diagnosis is that repeated examinations reveal the gradual increase in size of the sac.

(*b*) *Diagnosis of Rupture.*—(1) Early Rupture.—In all cases of early rupture the symptoms come on with remarkable suddenness. Generally the patient's last menstrual period has been somewhat delayed, and has then come on, but not normally, the character of the discharge being altered. She is probably going about apparently perfectly well, when she is suddenly seized with violent pain in the abdomen, becomes faint, and has to lie down. That this condition is not merely one of syncope is determined by the increasing pallor of the countenance, by the fact that acute abdominal pain is present, but especially by the fact, that whereas in syncope, when the pulse returns, it is slow and full, it is in this condition rapid, small, and thready. Indeed the patient presents all the symptoms of internal hæmorrhage more or less severe. Examination of the pelvis reveals very often little or nothing, but in some cases it is possible to detect a swelling in the Fallopian tube, and free fluid may be detected in the pouch of Douglas. Later, as coagulation takes place, a distinct boggy swelling is felt in the same situation.

(2) Later Rupture.—(*a*) Intra-peritoneal.—When this occurs the symptoms are similar to

those of early rupture, but very much more severe and alarming. The patient, either without any obvious cause, or after some exertion, is suddenly seized with excruciating pain in one or other side, becomes faint, and rapidly collapses. The surface of the body becomes pallid, the limbs cold, the pulse becomes feeble and rapid, and later imperceptible. Generally the patient remains conscious, but frequently suffers from nausea and vomiting. She may die in the course of a very few hours, and during this time the abdomen becomes more and more distended with blood. In some cases where the hæmorrhage is not so impetuous the patient may survive, the result being the formation of a large hæmatocele, but she is always liable to further hæmorrhage, and repetition of all the symptoms of the first attack, and death.

(β) Extra-peritoneal.—When the rupture takes place between the layers of the broad ligament the symptoms are similar, but not so urgent. Pain and faintness come on suddenly just as before, but actual collapse is not so marked, and the patient much more rapidly recovers from the shock. If the fœtus has died she may have no further trouble, if the hæmatoma is speedily absorbed, but she is exposed to the risks of suppuration, lithopœdion, etc. Unfortunately, in many cases the fœtus goes on developing, the broad ligament becomes more and more distended, until ultimately it gives way—secondary rupture—causing all the symptoms of severe intra-peritoneal hæmorrhage.

(*c*) *Diagnosis at Term.*—In those few cases in which the fœtus goes on developing to full term labour pains set in, and may continue for hours or for days. Generally there is hæmorrhage from the uterus, and a further discharge of decidual membrane. The diagnosis is arrived at by careful examination of the uterus or of the abdomen, in which the fœtus is found lying free. The fœtal heart sounds are usually distinct, and the mother may be conscious that the fœtus has been lying unduly to one side of the abdomen.

(*d*) *Diagnosis after Term.*—In tubo-abdominal pregnancy diagnosis after term when the child is dead is not as a rule difficult. The shape of the abdominal swelling is characteristic, and the detection of the fœtal parts while the uterus is found empty enables one to arrive at a definite diagnosis. On the other hand, when the pregnancy is tubo-ligamentary, and especially if it is posterior subperitoneo-abdominal, the diagnosis may be exceedingly obscure, and is more so if some time has elapsed since the death of the child. There are, of course, no fœtal movements nor fœtal heart sounds. The patient's breasts may show no signs of pregnancy, and menstruation may have returned. Indeed, the only sign is the fixed abdominal tumour, the relations and characters of which it may be impossible to determine. It is only by a most careful inquiry into the history of the case, eliciting the gradual enlargement of the abdomen during nine months, the history of rupture of the sac and of spurious labour at term, that a diagnosis can be arrived at.

Differential Diagnosis.—There are several conditions which simulate both in symptoms and in signs an ectopic gestation. Indeed, in some instances an absolute diagnosis at the moment is impossible, and it is only by repeated examination and continued observation that a definite conclusion is arrived at.

1. Pregnancy occurring in the rudimentary horn of a bicornuate uterus closely resembles extra-uterine pregnancy; and in this case the differential diagnosis is impossible. Kussmaul collected thirteen such cases, the majority of which had been reported as tubal pregnancies. If an exploratory incision is made, the anatomical relations will at once clear up the difficulty, for, as pointed out by Mauriceau, in cornual the round ligament is situated external to the gestation sac, whereas in tubal pregnancy it is situated on the uterine side of the sac. Pregnancy occurring in one horn of a completely bicornuate uterus may go to term and give rise to no abnormal symptoms whatever.

2. Retroflexion of the Gravid Uterus.—On superficial examination this may be mistaken for an ectopic gestation, especially if it has become fixed by adhesions. There may be sharp pains and symptoms of pelvic pressure very similar to those of the graver condition. However, a bimanual examination, and especially a bimanual rectal, will as a rule readily differentiate the two conditions.

3. Anteflexion of the Gravid Uterus.—Without doubt this condition may in some cases, especially in primiparæ, give rise to suspicion of ectopic gestation. The writer has seen many cases among primiparæ, who, certainly pregnant, suffered from severe pelvic pains during the first two or three months of pregnancy, and even from occasional hæmorrhages, and in whom it was difficult at first to exclude ectopic gestation. In these cases the cervix becomes so elongated as to simulate the uterus itself lying behind the gestation sac. Examination under chloroform, however, generally reveals the true nature of the case, and if even then there is doubt the patient may be kept under observation, and as pregnancy advances the diagnosis becomes easy.

4. Fibroid Tumour of the Uterus with Pregnancy.—A pregnant uterus which is the seat of a fibroid tumour may be mistaken for an ectopic gestation. In such cases examination under chloroform is generally necessary, when, on the hardness of the swelling, its intimate relation to the uterus, its change of position with the growth of the uterus, and on the fact that the tumour does not grow so rapidly as does a gestation sac, an accurate diagnosis must depend.

It may be very difficult to differentiate between an old-standing solidified hæmatocele and a myoma. In such cases much importance is to be attached to the history.

5. Ovarian Tumour with Pregnancy.—Ovarian tumours with and even sometimes without an intra-uterine pregnancy may sometimes cause confusion. This is especially the case if the patient suffers from attacks of pelvic pain. It is in these cases that the possibility of a twin pregnancy—one uterine and the other extra-uterine—must be considered. The diagnosis depends on the history and progress of the case and thorough examination under chloroform.

Even more difficult is it to distinguish between an extra-uterine gestation and an ovarian tumour which has burst or whose pedicle has become twisted. The symptoms being often exactly similar, a diagnosis may be impossible until the abdomen has been opened; otherwise, the diagnosis must depend on the history and the exact relations of the tumour to the uterus.

6. Tumours of the Fallopian Tube.—It is not remarkable that dilatation of the Fallopian tube from causes other than a growing ovum should simulate an extra-uterine gestation. The physical signs, especially of pyosalpinx, with which are frequently associated either amenorrhœa or uterine hæmorrhages, closely resemble in many cases those of a tubal pregnancy. Further, the symptoms of rupture of such a swelling may be easily mistaken for those of hæmatocele.

The absence of pyrexia cannot be relied upon for a diagnosis, as many cases of pyosalpinx have no febrile symptoms whatever. Much must depend on an accurate history of the case, which will frequently reveal a distinct cause of suppurative salpingitis.

7. Abortion.—Extra-uterine pregnancy may sometimes be mistaken for simple abortion or an incomplete abortion. History and symptoms are often similar, but a careful bimanual examination will easily clear up the diagnosis.

Treatment

The treatment of extra-uterine gestation may be conveniently considered under the following heads:—

1. Before rupture.
2. At the time of rupture.
3. After rupture.
4. After suppuration, etc., of the fœtus.

1. *Before Rupture.*—When the condition is recognised before rupture the only treatment which should be adopted is operative. The tube must be removed entire by abdominal section.

2. *At the Time of Rupture.*—In these cases it is often extremely difficult to determine whether or not to operate at once. In some cases it is better to adopt an expectant treatment owing to the very collapsed condition of the patient, and to operate later, if necessary, when the patient has to a certain extent recovered from the shock. Fortunately, in some such cases, especially when the rupture is extra-peritoneal, the hæmorrhage ceases and no operation is required at all.

The line of treatment to be adopted depends on a diagnosis, if this be possible, of whether the hæmorrhage is intra- or extra-peritoneal. It is in the former that as a rule immediate operation is indicated.

Although one would not go so far as Werth, who says that "ectopic gestation is a malignant new growth, and therefore should be removed by operation at every stage of its development," there is no doubt that the treatment of extra-uterine pregnancy is essentially surgical. It is doubtless true that some cases recover without any active interference whatever, but in the great majority immediate or later operative measures must be adopted to save the life of the patient. The question of when to operate is sometimes difficult to determine. For example, in the latter half of a tubo-abdominal or a tubo-ligamentary pregnancy, if there be no signs of immediate danger some surgeons prefer to delay operation until full term with the view of obtaining a living, viable child. Others go farther and wait till some time after the death of the child, in order that the placenta and surrounding tissues, having become shrunken and less vascular, may be more easily manipulated. Each case, however, must be decided on its own merits, and the decision arrived at will be largely influenced by attendant circumstances.

In cases which recover without operation the pregnancy is terminated by the primary hæmorrhage. The hæmorrhage from the uterus ceases, the pain disappears, and the swelling becomes no larger. Under these circumstances it may sometimes be well to give the patient a chance of cure by absorption. But in so doing one must realise that a natural cure is not always satisfactory. Absorption may necessitate weeks and even months of complete rest, and even then the patient may suffer from various pelvic disorders, caused by the formation of inflammatory adhesions, and resulting in displacement and distortion of the generative organs. Further, the fœtus may be of such an age that complete absorption is impossible, and although the patient may seem well even for years, she is always liable to the risks of suppuration in and around the fœtus.

In all cases where the symptoms of pain, collapse, or hæmorrhage are acute, operation should be undertaken without delay.

The Operation.—As this operation is usually undertaken in an emergency the usual preparations must be hurriedly made. The abdomen is opened in the middle line and the clots rapidly removed, and the ovarian and uterine arteries must be secured first of all by the fingers and then by forceps. The active hæmorrhage being

thus controlled, the remaining blood, both fluid and coagulated, is removed, all being carefully examined for traces of an embryo or a mole. If the pregnancy be an early one, say at the second month, the operation then consists of a simple salpingo-oophorectomy; but if later, when the placenta has been formed, the procedure becomes more complicated. The difficulty is the treatment of the placenta. If it be still firmly attached to the interior of the tube it is best to endeavour to control its blood-supply, to cut the cord close to its placental attachment, and to close the abdomen without drainage, which simply increases the danger of sepsis. In favourable cases the placenta atrophies and gives rise to no further trouble. If, owing to a previous rupture, the placenta has escaped and has become adherent to the intestines, it is still more dangerous to attempt its removal, which only sets up further and often incontrollable hæmorrhage.

Sometimes the placenta becomes easily detached during the course of the operation, and may be safely removed along with the fœtus. When it is impossible to completely stop the oozing in the pelvis, it may be necessary to pack the cavity with iodoform gauze after washing out the cavity of the abdomen with a sterilised solution. If the other tube is healthy it may be left intact, but if diseased it should certainly be removed.

3. *After Rupture.*—As a rule the surgeon is called only some time after rupture has taken place. If this be extra-peritoneal and has occurred during the first two months of pregnancy, no operation should be undertaken unless there is evidence that the fœtus is still alive, and that the gestation sac is developing, when the operation must be performed at once. The operation in the early months may not be much more difficult than that for the removal of a dilated tube, but there is a tendency for much greater hæmorrhage to occur, and many vessels have to be secured. In the later months the operation becomes extremely difficult. Adhesions are numerous, and must be dissected carefully apart, or must be ligatured and divided. The hæmorrhage is profuse, and the gestation sac must be completely enucleated. In many cases, however, this is impossible, and the oozing is so persistent that packing the cavity and drainage by iodoform gauze is the only means of satisfactorily dealing with the condition, the sac, if possible, being stitched to the abdominal wound. In many cases, however, after opening the abdomen it is found, owing to the numerous adhesions and the excessive vascularity of the sac, it is better to deal with it extra-peritoneally. The peritoneal cavity is closed, and then if the sac be situated low in the pelvis, and can be easily reached *per vaginam*, it should be opened through the vaginal roof, the cavity cleared out and packed and drained by iodoform gauze. If, on the other hand, the sac is situated higher up and comes into contact with the abdominal wall, it is better to make an extra-peritoneal opening into it, just above Poupart's ligament, and to wash it out and drain as before.

Vaginal Section.—There is no doubt that in cases undertaken at the time of intra-peritoneal rupture, when the hæmorrhage is excessive and diffuse, the best and most rapid method of reaching the bleeding vessels is by the abdominal route. But, in other cases when the hæmorrhage is localised, and in many cases of early extra-uterine pregnancy which have not ruptured, vaginal section is preferred by some operators. In cases where the pouch of Douglas is occupied by a limited hæmatocele, the operation of opening into it through the posterior vaginal fornix is extremely simple. An incision is made into the bulging tumour, the clots are gently cleared out, and the cavity irrigated. The limiting membrane of the hæmatocele is not in any way interfered with, and the cavity is drained by a tube or by iodoform gauze. It becomes less and less, till ultimately it is impossible to introduce a drain, and the opening into the vagina closes up.

In some cases, on passing the fingers through the vaginal incision into the abdominal cavity, not only can the swelling produced by the hæmatocele be felt, but also the rupture in the tube from which the blood has escaped can be detected. In such instances it is sometimes possible to bring the tube down to the vaginal opening, to ligature its uterine end, and so remove it. In other cases, however, one has to be contented with simply removing as much as possible of the ovum, and irrigating and draining the sac as before. Under these circumstances the hæmorrhage is often very troublesome and persistent, but by ligature, thorough douching, and packing, it can generally be controlled. If not, the abdomen must be opened suprapubically, and the bleeding points, which have been out of reach *per vaginam*, secured.

The cases in which an extra-uterine pregnancy after rupture may be dealt with by vaginal cœliotomy must be carefully selected; they are those in which the vagina is roomy, where the hæmorrhage is limited, and where the swelling is situated mainly behind the uterus.

Of recent years some operators have advocated anterior vaginal cœliotomy, opening into the abdominal cavity between the uterus and the bladder. The fundus of the uterus is drawn through the incision by means of volsella, and so the uterine end of the tube comes within reach and can be controlled, and no doubt an early unruptured tubal pregnancy can by this method be easily ligatured and removed. But, if rupture or hæmorrhage from the abdominal ostium has taken place, it is not so easy to satisfactorily deal with the hæmatocele by the

anterior incision as by the posterior, for thorough irrigation and drainage are impossible. Although this method has been adopted by some, the writer's experience is that, except in a very few cases in which posterior vaginal cœliotomy is possible, in nearly every case of extra-uterine gestation both before and after rupture the abdominal incision is infinitely preferable, as the exact situation and relations of the gestation sac and its surroundings can be more accurately determined and the condition dealt with more thoroughly.

4. *After the Fœtus has undergone various Changes.*—When the fœtus undergoes mummification, calcification, or saponification, it may give rise to no further trouble, and operation should be undertaken only if local or constitutional symptoms arise. If, on the other hand, suppuration takes place, the abscess must be opened at the most superficial point, and if already rupture of the abscess has occurred into the rectum, vagina, bladder, or externally, the fistula should be enlarged and all debris removed. These sinuses are very obstinate and not at all amenable to treatment.

Mortality.—The mortality from operation on cases of extra-uterine gestation is best illustrated by the following statistics :—In Mr. Tait's list (1888) he had 42 cases with 40 recoveries. Dührssen (1897) had 38 cases with 36 recoveries. Mayo Robson had 23 cases with 22 recoveries, and Taylor (1899) had 42 cases with 41 recoveries.

Repeated Extra-uterine Pregnancy.—Several cases of this have been recorded, and they seem to show that in some there is a distinct predisposition to extra-uterine gestation. A few authors hold that in every case where operation for ectopic gestation is performed, the second tube, even though healthy, should be removed; but since the chance of repetition is only about 4 per cent, this cannot appeal to most operators.

Ectopic Gestation, Recent Views.—Since 1900 a vast number of contributions have been made to our knowledge of ectopic gestation. With regard, for instance, to the *varieties* of ectopic gestation, progress falls to be recorded. The occurrence of *ovarian* pregnancy, suspected previously, was first conclusively demonstrated towards the close of 1899 by Dr. Catharine van Tussenbroek; and since then cases have been reported by Hastings Gilford, Croft, Anning and Littlewood, Mayo Robson, J. F. Thompson, Mendes de Leon and Holleman, H. W. Freund and Thomé, and J. K. Kelly and A. Louise M'Ilroy. From these observations it seems clear that an ovum may be impregnated within its Graafian follicle, and develop for some time entirely surrounded by ovarian tissue; the ovary responds to the stimulus of the fertilised ovum so far as its structure enables it, numerous blood-vessels and intervillous spaces being found, but decidual cells or cells resembling them are not always found. The fœtus generally dies early, but in Croft's case it was still alive at the third month of gestation. The occurrence of the *primary abdominal* variety of ectopic gestation cannot be said to be as yet fully established, although cases such as those of Valdagni (*Ginecologia*, ii. 164, 1905) and L. Guidi (*Clinica ostet.* viii. 147, 1906) would indicate that such a form really exists.

The investigation of the *tubal* variety of extra-uterine pregnancy has gone steadily on, and there is now available for reference and research a large literature on the subject of the anatomy and etiology of the pregnant tube. The profession is indebted to H. Russell Andrews for summarising nearly one hundred articles dealing with those aspects of the subject (*Journ. Obstet. Gynæc. Brit. Emp.* iii. p. 419, 1903; iv. 280, 1903; ix. 454, 1906). From these researches it appears that decidual reaction in the pregnant tube varies very considerably in amount, that the deep position of the ovum in the wall of the tube is due either to its passing into a diverticulum, or else to a malignant or pseudo-malignant action of the fœtal cells which enables them to burrow, and that rupture of the tube may be due to the burrowing powers of the fœtal cells, although it may also be caused by hæmorrhage and by contraction of the muscular wall of the tube. The vexed question of the etiology of tubal pregnancy remains vexed. The action of a diverticulum is possible, but not proven; "the ovum *may* be embedded in a diverticulum, but it is by no means proved that it *must* be so embedded"; salpingitis no doubt is common, but it seems to be going too far to ascribe all the changes seen in the tube-wall to a causative inflammation (some, at least, may be the effects of the pregnancy), and to postulate a symptomless salpingitis in childhood to explain them; and mechanical obstruction to, or hindrance of, the passage of the ovum through the tube seems to be still one of the more probable causes of tubal pregnancy.

The clinical observations of the past few years have made us better acquainted with a number of unusual complications and associations of ectopic gestation. The recurrence of tubal pregnancy in the same patient has now been noted in a large number of cases; Haig Ferguson (*Trans. Edin. Obstet. Soc.* xxiv. 37, 1899) brought the literature up to date, and since then numerous additions have been made to the list. It would seem to be fairly common, for in twenty-one cases Haultain noted it four times (*Journ. Obstet. Gynæc. Brit. Emp.* ix. 409, 1906). Allen (*Boston Med. Surg. Journ.* cliv. 654, 1906) has reported a second ectopic pregnancy in the same patient within a period of fourteen months. Pregnancy may be repeated in the *same* tube (Hofmeier, *Berlin. klin. Wchnschr.* xlii. 847, 1905; Goodall, *Brit.*

Med. Journ. i. p. 1272, 1906). Ectopic pregnancy may also occur in association with fibromyomata of the uterus, and F. E. Taylor has collected a small number of observations, including one reported for the first time (*Ibid.* ix. 412, 1906). Notwithstanding all our operative interference, extra-uterine fœtuses are still occasionally expelled in fragments by the rectum; but it should be added that this is now rare in civilised countries. I have in my possession some fœtal bones which a patient passed with almost no suffering from the bowel; she thought they were chicken bones, and supposed she had carelessly swallowed them! Apparently even a tubal pregnancy may go to the full term (Amos, *Ztschr. f. Geburtsh. u. Gynäk.* liv. p. 169), and sometimes the children of extra-uterine pregnancies may survive, although they are often malformed (F. von Winckel, *Über die Missbildungen von ektopisch entwickelten Früchten*, 1902). Simultaneous intra-uterine and extra-uterine gestation has been observed, twins may be found in the tube, and a double monster has been recorded in a tubal sac.

No great change has taken place in the treatment of ectopic gestation. Abdominal section is generally regarded as indicated so soon as the diagnosis is made. The most difficult cases are those at mid-term, when it becomes a matter of doubt as to whether the placenta should be removed at once or left *in situ* for a few days or altogether; such cases are generally mesometric, and it is a good rule that if the placenta is situated above the fœtus it should be removed, while if it is below it may be left (but if it be left, and suppuration occur in it, the sac will require to be reopened).

Ectopion. *See* Ectopia.

Ectro.—In compound words *ectro* (from the Gr. ἐκτρωτικός, relating to abortion) carries with it the significance of defect or failure; *ectrodactyly*, for instance, is a state of defective development of the fingers and toes, one or more of which may be absent; *ectromely* is a defective or malformed state of one or more limbs; and *ectrotic* means abortifacient. *See* Pregnancy, Intra-Uterine Diseases (*Congenital Amputations*); Teratology (*Malformations of the Limbs*).

Ectropion.—Ectropion (Gr. ἐκ, out, and τρέπειν, to turn) is a condition of eversion or outward turning of some part, such as an eyelid, and it may be congenital (*e.g.* in fœtal ichthyosis) or acquired (from paralysis, spasm, cicatricial contraction, etc.) *See* Eyelids, Affections of (*Defects in position and connection*); Leprosy (*Symptoms of Lepra Maculo-anæsthetica*); Mouth, Diseases of (*Deformities of the Lips*).

Ectylotic.—Having the power of removing warts (Gr. ἐκ, out, and τύλος, a swelling or callus). *See* Warts or Verrucæ.

Ectysterocyesis. *See* Ectopic Gestation.

Eczema.

See also Alopecia (*Varieties, Premature*); Children, Development and Clinical Examination of (*Symptoms of Teething*); Conjunctiva, Diseases of (*Exanthematous Conjunctivitis*); Dermatitis Repens (*Diagnosis*); Dermatitis Traumatica et Venenata (*Diagnosis*); Diabetes Mellitus (*Complications, The Skin*); Erythema (*Localised*); Foot and Mouth Disease (*Eczema Contagiosa*); Gout (*Irregular Gout, Cutaneous System*); Herpes (*Diagnosis*); Hydropathy (*Affections of the Skin*); Hypnotism (*Skin Diseases*); Nails, Affections of (*Diseases of the Skin, Eczema*); Nerves, Peripheral (*Injuries, Symptoms*); Otorrhœa (*Causes*); Pruritus (*Consequences*); Psoriasis (*Diagnosis*); Puerperium, Pathology (*Nipples, Eczema; Areola, Eczema*); Scrotum, Diseases (*Eczema*); Skin, Bacteriology of; Skin Diseases of the Tropics (*Dhobie Itch*); Trades, Dangerous (*Flax and Linen*); Umbilicus, Diseases of (*Eczema*); X-Rays (*Treatment of Eczema*).

Part I. General Pathology and Therapeutics

General Considerations.—Before we can proceed to determine even approximately the nature of eczema, we must consider its intimate connection with the skin as a free surface tissue. So close is this connection that we might briefly describe eczema as an inflammation of position. The skin is specially liable to injury from its great extension in space, and from its habitual contact with the unceasing perturbations of the outside world. From our present point of view the external surface of the body is essentially a double net, or sponge, of fine blood-vessels and delicate, sensitive nerve-threads surrounded and shielded from these external perturbations by

certain defensive structures. This protective mechanism, which plays a part of prime importance in the processes of eczema, consists of a fibrous meshwork covered externally by stratified epithelium. Whether this fibrous meshwork be dense or lax, and whether the epithelium is well formed or incoherent, are matters intimately connected with the liability of the individual to eczema. Daily observation teaches that any surface irritation is followed by a physiological thickening of the vascular net at the point of irritation. In other words, the irritation is followed in some cases immediately, in others after a short period of local anæmia, by blood fluxion to the menaced part. All eczemas are in the main serous inflammations. We have not only blood fluxion, but exudation of plasma into the injured epithelium; the process finally ending in the exfoliation, or throwing off, of the injured tissue. The skin exhibits a marked tendency to the production of localised inflammation, due to its fibrous stratified structure, which offers resistance on all sides to the escape of the exudation. It is the resistance offered by the layers of flattened horn cells to the free exit of the plasma which leads to the formation of vesicles and bullæ. As we shall see later, this external resistance disappears under certain circumstances. In such cases there is a free exit for the plasma, and vesicles are no longer formed.

The manifestations of eczema at any particular spot, at any particular time, or in any individual case, vary according to the following circumstances: (1) the pressure of the plasma exudate, and the degree of resistance offered to its escape to the free surface; (2) the position or region affected; (3) on the nature and extent of the injury to the epithelium; (4) the resistive power with which the skin can oppose the irritant. Each of these circumstances must be carefully considered.

The Pressure of the Plasma Exudate and the Degree of Resistance offered to its Escape to the Free Surface.—The pressure of the plasma is proportionate to its bulk. The quantity of exudate is closely connected with the nature of the irritant and the resistive state of the cutaneous tissues. From the clinical point of view it determines the so-called lesions of eczema. The low-pressure eczemas are dry, red patches with a strong tendency to localisation and delimitation. If the pressure rises a few degrees the exudate is sucked up by the epithelial cells, forming a thickening known as the *papule*. A still higher rise manifests itself as a droplet of plasma under the horny layer (technically known as a *vesicle*). In people of middle or later life the resistance of the horn cells below the knees is often so defective that the exudate, instead of being temporarily confined in the form of vesicles, oozes directly on to the free surface (eczema rubrum). In regions where the dermal mesh is loose and open, as in the near neighbourhood of the eyes or in the scrotum or vulva, the exudate finds readier access to the lymphatics than to the free surface, and thus produces the general swelling which is so characteristic of eczema in these regions. Special resistance is offered to the escape of the plasma in the palms and soles owing to the density of the horny epithelium. In these regions the exudate is apt to be retained for lengthy periods in the form of vesicles. The accumulation and effort to escape may be accompanied by a sensation of burning and intense itching, in some cases even by a certain amount of constitutional disturbance.

The Position or Region affected.—The skin in the extended relations which it is called upon to meet varies appreciably in structure and function in different regions. In some localities the sebaceous function is predominant (face, scalp, chest, back, prepuce, ears, meati), in others the formation of hair, while certain regions are characterised by the size, quantity, and activity of the sudoriferous glands (palms, soles, axillæ, bends of elbows, and knees). These anatomical factors are certainly capable of modifying the processes of eczema. The presence of large quantities of fat tends, as Unna has pointed out, to restrain the serous discharge in response to injuries in these regions. The excessive outpouring of sweat, by diminishing the inertia of the surface, increases the liability of the skin to injury and inflammatory reaction. The presence of hair in large quantity, while a security up to a certain point against injury, and therefore against serous inflammations, greatly retards recovery on account of the hindrances which it offers to the dispersion of the plasma. From the readiness of access which inflamed hair-follicles present to pyogenic organisms, the exudate in such regions is liable to become bacteriologically infected. Hence the purulent nature of the plasma so commonly found in eczema in hairy regions.

On the Extent and Nature of Injury to the Epithelium.—The full discussion of this will be taken under the head of *Etiology*. Here I would simply point out the broad generalisation that the injury is the resultant between the irritant force on the one hand and the resistive force of the skin on the other. In some persons the skin is capable of resisting gross insults, while in others, with weak resistive power, comparatively slight irritants may result in severe injury.

The Resistive Power of the Skin.—There is no more important cause in the development of eczema and in the modification of its manifestations than the total resistive power which it presents to irritants. This resistive power is the sum of many forces which we may conveniently con-

sider under the two heads of passive resistance and vital resistance. The passive resistance of the skin, to which reference has just been made, is afforded by the keratin and fat cells of the epithelium, and by the underlying fibrous meshwork. Regions of the body which are habitually exposed to pressure, such as the palms, soles, and buttocks, are provided with a thicker armour of horn cells; flexible regions, where great mobility is required, such as the groins and axillæ, the elbows, the bends of the knees and the palms, are rich in fat. The dermal felt-work is mainly responsible for resisting pressure and mechanical blows. In infants, on account of the extreme delicacy of the epithelium, it is the main defence of the skin, and its imperfect state of development accounts to some extent for the proneness of young children to eczema. Vital resistance is afforded by the so-called trophic and vaso-motor nerves. We shall have occasion to refer again to this.

The Lesions of Eczema.—It is the custom to divide the eczematous eruptions into four groups, according as the predominant lesion is papular, vesicular, pustular, or erythematous. Although this method has received the sanction of some eminent writers on Dermatology, and has been blindly followed by others, it is not one which commands universal approval. The minute description of lesions only serves to draw the mind away from the essential nature of eczema. The so-called lesions of eczema have no separate basis of existence. They are mobile, interchangeable marks, due either to the temporary retention of the exudate within the epithelium (vesicles, bullæ), or to the addition of leucocytes to the plasma (pustules). The essential thing to remember is that all the papular and dry discoid forms are essentially low-pressure eczemas, while all the vesicular and sero-purulent varieties are high-pressure eczemas. The histological differences between these several forms are very slight, as Unna and Leloir have so well shown. The vital circumstance which directs the inflammatory manifestation into one or other of these forms is the physiological state of the skin, which determines the amount of resistance which it shall offer to the irritant which is the cause of the injury.

In view of these considerations our description of the lesional varieties will be no longer than is just necessary to enable the reader to recognise them when seen.

The Vesicle.—The eczematous vesicle may be defined as the temporary retention in the superficial part of the epithelium (usually immediately under the horny layer) of a drop of serous plasma derived from the capillaries. For diagnostic purposes we have to distinguish it from the vesicles of zona, and from those of the herpetoid eruptions which in some cases follow nerve injuries. The eczematous vesicle is a flat elevation, when much resistance is offered to the escape of the plasma, or a conical or dome-shaped elevation when the resistance is less. In herpes zoster the outline of the vesicles is corymbiform, and their development and arrangement are determined by the peripheral distribution of the irritated nerve fibres. The herpetoid vesicles of certain nerve injuries, described by Charcot, Weir Mitchell, and others, are small, scattered, acutely pointed elevations filled with clear serous fluid. From the fact that they tend to leave scars we may infer that they are the result of a colliquative process, like those of zona, and not a manifestation of a general serous inflammation. The vesicles of cheiropompholyx, which some authorities regard as a form of eczema, are characterised by the depth of their position and by a curious translucency, which gives to them the aspect of boiled sago grains deeply buried in the skin. The presence of vesicles in large numbers in the course of eczema is to be taken as evidence of a high degree of irritation with (usually) diminished resistance of the skin. With the rupture of the vesicle the serous albuminous exudate escapes to the free surface, where it coagulates or "*scabs.*" The period occupied by the exudate in gaining free access to the open surface is, in some cases, marked by intense burning and itching. But these sensations vary in intensity according to the degree of sensitiveness of the patient's nerves. (For treatment, see Clinical Types of Eczema, p. 36.)

The *pustule* of eczema is a vesicle to which leucocytes have been added in numbers sufficient to render it opaque to light. When pustules are the predominant lesion in eczema it may be taken for granted that some special element has entered into the causation of the inflammation. In many cases this is due to the impressionableness and sensitiveness of the cells of the skin and blood, as in infants and young children, in others to a greatly impaired resistive power of the skin, as in weakly debilitated people. Frequently it is due to the invasion by pyogenic organisms. In rare instances the organisms are chromogenic, when the pustules assume a special colour, orange or blue, according to the organisms. Pustulation is common in hairy regions, such as the scalp and face, owing to the favourable conditions for development of microbes afforded by the succulent follicles.

The Papule.—This lesion may be clinically defined as a minute, rounded, conical, dry elevation of the epithelium. According to Unna it is a little hypertrophic, œdematous thickening of the prickle-cell epithelium. When the papule is the predominant lesion of eczema it is an indication, as we already stated, of the low pressure of the exudate. The drier and more solid the papule, in a word, the nearer it approaches to the true lichen papule, the more

difficult is its dispersion. The reason of this is that we are not dealing with a simple serous exudate temporarily retained, but with a neoplastic process. The lesions are specially liable to mechanical injury (scratching) from the severe itching which accompanies their development. In this way a serous inflammation may arise out of a dry papular eczema.

Dry Discoid Patch-lesions.—This form of lesion is met with in the seborrhœic eczemas. Like the papular lesion they indicate low-pressure exudation, and show a marked tendency to circumscribed limitation of the exudate. The patches tend to fade in the centre. The discharge, which is commonly small in quantity, is remarkable for the large admixture of fat.

The *Erythematous* Variety.—In this form vascular paralysis and œdema are the most predominant features. The exudate, instead of travelling to the free surface, gains access to the lymph and juice spaces, owing to the lax conditions of the dermal mesh. In consequence of this the surface may be hot, red, and swollen, but dry. It is liable to injury from scratching, and is in many cases accompanied by maddening irritation.

Etiology and Classification.—To rightly estimate the various manifestations of eczema with a view to their causation and classification, two orders of facts must be kept clearly apart: 1st, those appertaining to the actual injury with its inflammatory reaction; and, 2nd, those which refer to the liability of the skin to injury. Broadly speaking, eczema is always related to some surface [1] injury, although not necessarily to one of an ostensible kind. In not a few cases we can identify and name the irritant. It may be iodoform, mercury, borax, or some vegetable poison, such as primula obconica or rhus toxicodendron. But in the vast majority of instances we are completely ignorant of the exact nature of the irritant. I am not sure that the omission matters much. The irritants, whatever they may be, are only relative irritants. For with the possible exception of the mycotic forms, nothing is better proven than that no substance has an absolute specific relation to eczema. To establish a classification of the eczemas from the standpoint of the irritant is not at present possible, and I cannot imagine that it ever will be. But it is fairly within our power to arrange the various eczematous forms on certain broad principles as follows:—

Class I. Accidental Eczemas (trade and traumatic dermatites): forms in which the irritant is the dominant and determining factor in the process.

Class II. Intrinsic Eczemas (eczema vulgaris): forms in which the surface-making tissues are the dominant and determining factor in the process.

Class III. Mycosiform Eczemas (seborrhœic eczema, Unna; seborrhœa corporis, Duhring): forms which are associated with the growth and multiplication of certain vegetable micro-organisms, and which commonly occur in fatty or seborrhœic skins.

We have now to consider the etiology of these several classes.

I. *Accidental Eczemas.*—For the full list of external agents capable of producing eczematous eruptions the reader must consult the article on "Artificial Eruptions." They belong to the four groups,—animal, vegetable, mineral, and atmospheric (cold, heat, solar rays, X-rays, electricity).

The effect of a surface irritant varies with (1) the nature of the irritant, (2) the mode of its application, (3) the duration of its action, and (4) the resistive power of the skin.

(1) *Nature of the Irritant.*—Irritants may be (1) *absolute*, such as caustics; (2) *essential*, such as freezing, the result following, not immediately, but after the lapse of some time; or (3) *relative*, such as light, which requires a very low degree of resistive power before it can become an irritant. The intensity of a surface irritant is its own force minus the resistive force of the skin. The intensity is therefore purely relative to the surface tissues of the person affected. Consequently, in the class of accidental eczemas we cannot altogether exclude the influence of the tissues themselves. No classification can be based on the nature of the irritant. Experimental pathology has shown that the attempt to mark off sharply inflammations caused by mechanical and chemical noxæ from those produced by bacteria must be given up (Adami).

(2) *Mode of Application.*—The irritant may be directly conveyed into the skin by a sharp instrument, such as a lancet, or by the prick of a thorn. It may pervade the tissues of the skin, as in the case of ethereal waves of light. It may be held in contact with the skin by means of clothes. The application may be medicinal or cosmetic, or it may be applied deliberately for illegal or immoral purposes.

(3) *Duration of Action.*—Excluding agents which directly and instantaneously kill the epidermis, all irritants require the lapse of more or less time to produce an inflammatory reaction. The trade or professional eruptions are in some cases the accumulative result of innumerable stimuli of moderate intensity, the workman remaining to all appearances unaffected till circumstances lower the resistive power of the surface tissues.

(4) *The Resistive Power of the Skin.*—We may obtain every degree of inflammatory reaction from hyperæmia up to gangrene, according to the resistance offered to the irritant by the sur-

[1] By the word "surface" is implied all those tissues which are directly concerned with the making and the maintenance of the free surface, on whose integrity and inertness the health of the skin depends.

face tissues. Many handicrafts are in themselves injurious, and their daily pursuit must be accompanied by a process of slow attenuation of the resistive power of the skin. A good example of the part played by this resistive power in the face of irritants is the common flannel rash. Rough flannel will not produce a papular eczema in a person whose skin is dry, firmly coherent, and innervated by quiet nerves; but the same flannel worn by the same person when the skin is softened and macerated by excessive sweating, and when the cutaneous nerves are irritable, is sufficient irritation to produce the rash.

II. *Intrinsic Eczemas.*—This class includes the commonest and in many respects the most important forms of eczema. They are to be etiologically distinguished from the eczemas of Classes I. and III. by the fact that the particular irritant exercises very little control over the inflammatory manifestation, this being mainly determined by the diminished power of resistance on the part of the surface-making tissues. We must fix our attention, therefore, on the epithelium, with the underlying double net of capillaries and nerves. It is here that these eczemas are developed. It is difficult to see how any interior organ, or functional disturbance, can have the slightest influence on the origin and maintenance of eczema unless it affects the equilibrium of the surface tissues. I must again repeat that of *definite morbiferous particles* capable of producing these intrinsic eczemas we know absolutely nothing. It is enough for practical purposes to assume that some subtle injury has been inflicted on the epithelium. But we have now to consider those circumstances which increase or diminish the liability of the skin to injury. We may pursue this inquiry in two directions—1st, What are the forces which habitually tend to inhibit or restrain the inflammatory manifestation? 2nd, What are the circumstances which influence this power of resistance?

When we consider how large is the quantity of blood contained at high pressure within the capillaries, arterioles, and venous channels of the skin, and that their only protection from the atmosphere is afforded by a delicate nervous membrane, we can understand the need for some mechanism of control. In addition to the passive resistance of the fibrous mesh, the inert horny cuticle, and the fat which is so abundantly distributed through the epithelium, there exists an elaborate nervous mechanism for the purpose of restraining the motor excitability of the arterioles and capillaries, and thereby maintaining the cohesion of the blood-stream and the regularity of its flow. The innervation of the skin is discussed in the article on the anatomy and physiology of the skin, and it will suffice to say here that the evidence points clearly to two sources of control, namely, the brain cortex and the vaso-motor mechanism of the skin itself.

The physiological experiments of Goltz and Ewald on the dog, of Sherrington on the monkey, and of Saviotti and others on the frog, have clearly demonstrated that transection or removal of the spinal cord does not produce any permanent alteration in the circulation through the skin, all the functions of which are regularly maintained so long as the animal is protected from injury. Clinical experience is quite in accord with these physiological results. The extended experience of Drs. Weir Mitchell, Moorhouse, and Keen, on gunshot injuries of nerves, has shown that the complete cutting off of a limb from central nerve influence is not followed by loss of blood tension in the isolated limb, the cohesion of the blood being still maintained. But since the time of Cohnheim's experiments on the cornea, and onwards, it has been a matter of common observation that loss of sensation in the skin greatly increases its liability to injuiry, and therefore to inflammation. In Professor Sherrington's experiments on the monkey, in which the spinal cord had been divided in the cervical region, he frequently observed ulceration over the external malleolus, which he attributed, not to any trophic disturbance, but to pressure during the paralysed condition of the limb. The inference to be drawn from these and many other facts of similar import is that the skin receives a considerable share of its power of resistance from the cortex of the brain, but when this source is cut off there still remains a local mechanism in the skin itself which is capable of maintaining the cohesion and tension of the blood. Further, it may be inferred that any block to those impulses which are constantly descending from the cortex to the periphery, or to those which are constantly ascending from the skin to the brain, greatly increases the liability of the skin to injury, and therefore to serous inflammation.

It is a pertinent question whether this highly important and demonstrated fact of the independent nervous control of the skin may enable us to view the origin of the intrinsic eczema in a somewhat new light. In the light of this fact it is not unreasonable to assume that eczema is not something which is "caught" or implanted in the skin, or the elimination of some poison through the cutaneous tissues, but, on the contrary, something the elements of which are present in all of us, and which are only kept down by certain natural checks. According to this view eczema is inevitable in all cases where persons have lost their natural securities. If this be the true and right way to regard eczema, as I think it is, then, so far from being an entity, it is simply *skin*, or a portion of skin, temporarily deprived of its natural checks to inflammation. These checks or securities withdrawn, the skin inflames, or becomes eczematous, just as water boils when atmospheric pressure is withdrawn, namely, by

its own inherent expansive power, and not by reason of anything which is added to the water.

We have now to consider the circumstances which influence the resistive power, or, in other words, the nervous control of the skin.

1. *Age and sex* affect the origin and development of eczema only so far as they modify the physiological state of the skin. The skin, like the cortex of the brain, gradually acquires the power of control, passing through the immaturity of infancy, the revolutionary and transitional state of adolescence, the maturity of manhood, and, finally, the senility of old age. The control over the capillaries and arterioles varies in each of these periods, being defective during infancy, unstable during adolescence, forcible during manhood, and weak but stable during old age. On the evidence of these physiological facts alone we should be led to conclude that the intrinsic eczemas are especially prevalent during early childhood, common in adolescence, less so during manhood, and still less frequent in old age. The evidence of statistics of hospital and private skin clinics in large cities is much in accordance with this *a priori* reasoning. There is, however, one apparent contradiction, for clinical experience undoubtedly reveals a notable increase in the proportion of cases of eczema during adult life, at the very time when we should have supposed the neuro-vascular control to have been greatest. But this apparent exception is in reality an additional proof of the conclusions arrived at above. Taking Dr. Bulkley's statistics of 5216 cases of eczema, we find in his private practice the proportion of cases occurring between the ages of thirty and fifty to be 501 males and 265 females, while in his hospital clinic the ratio for the same period was 372 males and 431 females. The inference to be drawn from these facts, which agree in the main with the experience of other dermatologists, is that the prevalence of eczema during adult life is due partly to the abuse of the nerve-controlling apparatus which the struggle for existence entails, but also to the greatly extended relations of the men and women who have to purchase their existence at the price of incessant daily toil. Thus, while it is physiologically true that the nervous control is at its height during manhood, it is also true that the irritation and dangers to which the skin is liable have enormously increased.

2. *Inherited Variations in the Neurility of the Skin.*—The term *neurility*, which was introduced by Lewes and adopted by Vulpian to express the property of nerves to be excited, I employ here to denote the irritability of the combined neuro-vascular apparatus of the skin. No fact has more impressed itself on my mind during my professional career than the wide variations in the character of the human skin. It seems, like the brain cortex, to have an individuality of its own. Indeed the skin and brain cortex often share certain characteristics in common. Nor is this to be wondered at when we call to remembrance the fact that, from an embryological standpoint, the brain is an offshoot of the cutaneous epithelium. There is a type of skin in which it is difficult to obtain a neuro-vascular reaction by external irritation, in which the neuro-vascular apparatus pays, as it were, very little attention to the perturbations which are transpiring around it. We can handle these skins, therapeutically, with freedom, and apply powerful stimulants with little or no risk of provoking a serous inflammation. This type of a quiet skin may suffer from psoriasis, rarely from moist eczema.

A type of a very different sort is afforded by persons of a predominantly nervous character, or with a strongly developed tendency to arthritic rheumatism. Such persons carry about with them a sponge-like neuro-vascular apparatus which feels and moves with every external and internal perturbation. Individuals with hypersensory-motor skins of this type are ever liable to catarrh, because they are always too much exposed to injury. If they drive in the wind, or sit in the sunshine, or swim in the sea on a sunny day, they are apt to develop an irritable eczema. We may cure the eruption over and over again, but not the tendency; that only yields to thinning of the cortex, or to the dulling of nerve sensibility, the consequence of old age.

Another variation in the neurility of the skin, and in its way one no less characteristic, is met with in scrofulous young persons. This character is to the skin what melancholia is to the "mental cortex," and essentially consists in the slow rate of change from one capillary state to another. It is in skins of this type that we so frequently find chronic rebellious, circumscribed patches of eczema, especially in the neighbourhood of the eyes, nose, mouth, and ears in young persons.

3. *Acquired Variations in the Neurility of the Skin which increase its Liability to Surface Injury.*—That the skin may pass from the normal stable condition into one less stable and less able to resist injury, is a fact amply supported by clinical observations, although the steps by which this degeneration takes place are not yet discovered. When eczema is seen to follow some outstanding pathological or physiological event, such as mental shock, pregnancy, a fit of the gout, the cutting of teeth, gastrointestinal irritation, nephritis, etc., the constitutional disorder is dogmatically affirmed to be the "cause" of the eczema. Many such cases have been collected together by Dr. Bulkley of New York in a valuable paper. He records and cites some remarkable cases following psychical shock, reflex nerve disturbance from the mouth (teeth),

intestines (tape worm), uterus (pregnancy), urethra (stricture), and eyes (after straining). To rightly estimate the importance of these constitutional disturbances we must remind ourselves of the fact that their concurrence with eczema is not the rule, but quite exceptional. How many cases of eczema do we see in which no notable constitutional disturbance of any sort is present, for one case which presents some striking constitutional event or abnormality? The ratio doubtless varies according to the class from which we draw our patients. In cities such as New York or London, where the rush and tear of life is a formidable factor of civilisation, nerve exhaustion and nerve disorders must of necessity be more frequently met with in association with eczema than in quiet rural populations. The right conclusion seems to be that any pathological or physiological event which lowers the cutaneous vaso-motor control, which renders the capillaries more excitable, which diminishes the cohesion of the blood, which increases the permeability of the capillary walls, which over-stimulates the epithelium, does certainly increase the liability of the skin to injury, and therefore to serous inflammation. No circumstance whatever, in the opinion of the present writer, can convert eczema into a true constitutional disease. However it may arise, it is essentially a local process—a local expurgation or throwing off of injured surface tissues. Take away the natural securities of the skin, diminish the local control below a certain point, and the ordinary perturbations of common life are quite sufficient to produce eczema.

4. *Relations of Eczema to Gout.*—The popular notion that eczema is very frequently of gouty origin is open to grave criticism. Not only is there no proof that the uric acid compounds can directly originate eczema, but the evidence, so far as it goes, points to the opposite conclusion. Gout is rare under twenty years of age, but the formation of uric acid is known to be greatest in early life. Recent researches have shown that uric acid may be present in the blood without the development of gout (see "Gout"). Sir Alfred Garrod has pointed out that the failure on the part of the kidneys to eliminate uric acid is not accompanied by a vicarious excretion of this principle by the skin. But the most direct proof of all is the tolerance of the skin for uratic deposits. The formation of tophi in the external ear, and in the neighbourhood of the joints, is not accompanied or followed by an eczematous eruption. On the other hand, it cannot be denied that gouty persons are prone to eczema. In the experience of Sir Alfred Garrod about 30 per cent of gouty subjects suffer from eczema. In the opinion of the present writer it is not the uric acid, but the nerves of the gouty man which predispose him to eczema. That inherited type of peculiar tissue metabolism which culminates in gout is accompanied by an attenuation of the resistive power of the skin, which makes the patient less able to resist the effects of surface injury. There is nothing to distinguish gouty eczema objectively from many other forms of intrinsic eczema. It is usually confined to localities where the capillary circulation is below the average velocity, or where it is apt to be interfered with, and which, from their exposed position, are specially liable to injury, such, for example, as the face, ears, hands and feet, flexures and genital region.

III. *Mycosiform Eczemas.*—The hypothesis that eczema is of parasitic origin finds its chief advocate in Unna, who goes so far as to assert that the disease is a uniform one both as regards its histology and etiology. It would serve no useful purpose to discuss this difficult matter in the present imperfect state of our knowledge. I shall, therefore, content myself with a brief statement of the facts which have been added to our stock of information within recent years. There is good ground for believing (1) that several forms of living cryptogamic vegetations exist in the serous and fatty exudate of the mycosiform eczemas; (2) that the microbic forms at present isolated are the bottled-shaped bacillus, the micro-bacillus, and the diplococcus of Unna (morococcus); (3) that the morococcus is allied, morphologically and biologically, to the staphylococcus albus, and that it is capable of producing impetigo-like vesicles when experimentally inoculated into the human skin; (4) that the morococcus is very prevalent in some forms of seborrhœic eczema, but scanty in others. The exact pathogeneity and bacteriological position of these organisms have not yet been determined, so that a bacteriological classification of the mycosiform eczemas is impossible in the present state of our knowledge.

The bottle-shaped bacillus seems to bear no definite relation to eczema or any other cutaneous disease. They are seldom absent from moist fatty horn cells. We may infer from this that they are innocent saprophytes. The vegetations of the "micro-bacillus," an organism isolated and described by Sabouraud, are equally abundant in seborrhœa capitis and in alopecia areata, and the same bacillus is present also in the comedo of acne. Clearly it is attracted by a fatty soil, but neither its relation to seborrhœa nor the scope of its morbiferous actions is yet defined. The cocci which Unna has found in the scales and fatty discharge in so many cases of seborrhœic eczema show no very distinctive characters under the microscope. In the drier and more superficial portions of the exudate they cling together in mulberry-like masses, a feature in their character which Unna has sought to perpetuate by naming them *morococci.* All that we know at present about these organisms points to their affinity with the staphylococcic group. Their sudden entrance into the prickle layer of the epithelium

provokes a seroleucocytosis. A thinner and more watery pustule forms than in ordinary impetigo. Nevertheless these so-called acute eczema vesicles of Unna are probably, as Török maintains, of the nature of impetigo, and not true eczematous vesicles. Unna himself recognises their impetiginoid character, and says they are not to be confused with those secondary eczematous vesicles which are part of a general serous inflammation. Unna, however, is not content with pointing out the prevalence of these cocci in the mycosiform eczemas, but claims for them the power of origination, and regards their presence as the final proof of the identity of eczema. Considering the imperfect nature of the evidence we must regard these opinions as hypothetical and non-proven. Notwithstanding these bacteriological difficulties we are encouraged to hold on to the parasitic theory from considerations relating to the course and development of this particular class of eczemas. Unna has rightly pointed out the prime importance of sterilising the surface of the scalp, and of correcting the seborrhœa in order to cure these eczemas, the scalp being regarded as the fount and origin of the inflammation. Further support of the hypothesis is afforded by the fact that local remedies which have the effect of killing the injured epithelium, destroy at the same time its infectivity, and arrest the centrifugal extension of the inflamed patches.

Histo-pathology.—It is a curious fact that the naked eye is capable of discerning many variations and distinctions in the lesions of eczema which disappear under the microscope. As Unna remarks, so simple and uniform are the structural changes of eczema that it is difficult to avoid the conclusion that we are dealing with a uniform *histological* process. Nor is there anything in this notion which contradicts the belief that eczema, regarded from an all-round point of view, is a disease of different kinds in different persons. It is the individual that determines what kind, and not the histological process. From a precise pathological standpoint, eczema may be defined as *a device to throw off an injured epithelium.* The method by which this is effected is essentially the same in all varieties of eczema, both acute and chronic, and consists of:—

(1) Blood fluxion to the menaced or injured part.

(2) Exudation of plasma into the epithelium.

(3) The softening and loosening of the epithelium in consequence of this exudation.

(4) The stimulation of the deep germinal epithelium to new growth.

(5) Encapsulation of the softened œdematous epithelium by an undergrowth of impervious horny tissue.

(6) The casting off of the injured portion.

(7) The contraction of the capillaries, and the restoration of vaso-motor control.

The microscopic changes in the skin are as follows:—

1. *Epithelium.* — Cellular and intercellular œdema, the degree depending on the acuteness of the process. Broadening of the plate-epithelium, and deepening of the cones. If exudation is excessive, or if external pressure resists the escape, a split occurs between the horny and granular layer to accommodate the plasma (vesicle). The horny external layer is converted into a soft nucleated membrane. With this softening of the surface one of the main defences of the skin against external irritants is removed. This softening and nucleation of the surface horn cells is called *parakeratosis,* in contradistinction to keratosis, which is the normal process of surface induration and the disappearance of the nuclei and cell-contents. In dry eczema, especially in the papular forms, the growth of epithelium is more pronounced than in the moist varieties, while the spongy condition is less marked.

2. *Papillary Body.*—The shape of the papillæ is modified according to the downward growth of the cones. Capillaries are dilated. In some acute cases there is a gathering of leucocytes in this situation, but they may be normal or subnormal in quantity.

3. *Derma.*—The connective tissue corpuscles are greatly increased in number, with increase of collogen, in cases of indurated chronic eczema. Leucocytary infiltration is absent in the true eczemas, this fact serving to distinguish them from the dermatites.

4. *Leucocytes.*—The exact part played by the different varieties of leucocytes in the eczemas has not yet been determined. Observation points to the conclusion that they are only of secondary importance. Their presence in large numbers is to be taken as an indication of some additional element in the etiology, such as laxity and debility of tissue, the invasion of the skin by pyogenic organisms, etc.

5. *Ferments* play, probably, a not unimportant part in the pathology of eczema. The present writer has shown that irritated human epithelium produces, under certain circumstances, a proteolytic ferment capable of converting the proteids of inflammatory plasma into peptones, and of liquefying gelatine outside the body. The fibrin ferment is probably derived from the spitting up of leucocytes, or of certain leucocytes.

6. *Nerves.* — There is almost a complete absence of exact information regarding the state of the cutaneous nerves in acute and chronic eczema. Twenty years ago (1879) Colomiatti published his histological observations of a number of eczemas of papular squamous type. He found "essential changes in the cutaneous nerves." Leloir's efforts to confirm Colomiatti's observations at first failed; he could find no evidence of disease either in the nerve-fibres or in their sheaths. But in his

later researches he states that he is able to confirm Colomiatti's observations. Since then no further evidence has been adduced, although competent authorities have searched. The inference to be drawn seems to be that no structural lesion of cutaneous nerves underlie the development of eczema, but that in certain rare instances the nerve-fibres may be involved in the inflammatory process.

The Principles of the Treatment of Eczema

I. *Eczema is cured by Local Expurgation.*—All successful treatment of eczema is based on this method, and the more penetrating our insight into the working of this process the more successful are our efforts likely to be. When our methods of treatment fit into the natural process of recovery, the result is a quicker cure; but when there is no sort of harmony between them, the consequence of our interference is a delayed cure or an aggravation of the original disorder. Considering the prime importance of this principle, it is well worth our close attention. It has already been pointed out that the essential act of eczema is the discharge of plasma into the epithelium, with the subsequent encapsulation and casting off of the injured portion. This is the whole intent of eczema as a pathological process. Striking proof of this is afforded by the fact that the subsidence of the inflammatory symptoms begins with the growth of a horny impervious membrane *under* the œdematous and infective portion of the epithelium, thereby completely excluding the injured from the uninjured portion of the epithelium. As long as the infected œdematous portion of the surface remains organically connected with the rest of the epithelium, so long will the eczema persist. If nature could, unaided, effect a spontaneous cure, it would be by the throwing off of this injured portion, and if we have any therapeutic control over eczema it is only so far as we can aid this expurgation.

Hence the treatment of eczema resolves itself into a definite piece of work in dynamics. It is a pertinent question why the dynamics of nature are so defective as regards the spontaneous cure of eczema. In other words, what are the causes of the chronicity of eczema? Briefly, I believe the explanation to be that when vaso-motor control is once lost, and the surface has become soft and œdematous, that the perpetual irritation of the outside world prevents the surface tissues from returning to their normal state of health. But, further, there is a strong tendency in the skin to stagnation outside the blood-currents of the cutis. Thus leucocytes and micro-organisms carried from the blood-stream into the epithelium degenerate and die; and plasma in the same situation tends to stagnate, as is frequently observed in the length of time that epithelial vesicles may remain unchanged.

Since the success of our treatment depends on the harmony between it and the spontaneous curative methods of nature, it is of prime importance to the medical adviser to note precisely the state in which the eczema is at the moment when he is called to interfere. He will find the eczema to be in one of the following states:—

1. *The plasma exudes at high pressure; the surface is moist and defective in covering.* In this state all we can do is to anticipate the next stage of drying or desiccation. We can render valuable service in this direction by the employment of astringent lotions, such as weak solutions of silver nitrate in distilled water. If the tissues are excessively œdematous, and the discharge thick and purulent, then desiccation is better attained by the continuous application of Lassar's paste with 1 or 2 per cent of salicylic acid (*see* p. 33).

2. *The plasma does not exude at high pressure, but the surface is œdematous, while the drying process, evidenced by scabbing, has begun.* Here again our remedies must be chosen with a view to aid the drying process. Care must be taken to remove all scabs, as they are infective and contain irritative organisms. An antiseptic but non-stimulating porous ointment should be selected. Salicylic acid not exceeding 2 per cent is perhaps the safest and best of remedies in this stage.

3. *All active exudation has ceased, a new surface has formed, but the whole of the surface tissues are soft and still contain an excess of lymph and plasma.* As this first formation of a new surface is never stable or permanent, we can again anticipate nature by removing it. This may be effected by the employment of a gentle irritant. Since it is advisable not to increase the heat of the part, porous ointments are better than simple fatty ones. In many cases calamine-zinc lotion, with the addition of 1 per cent boric acid, or liquor picis carbonis, answers admirably. It has the great advantage of being drying, protective, and stimulating at the same time.

4. *There is no exudation; the surface is dry and rough, the process of expurgation having been arrested.* It is now that the real work has to be done, namely, the separation of the injured surface, and the dispersion of the stagnant plasma and cells. To effect this we must employ irritants, and often in strong proportions, such as sulphur and tar (ʒj. or more to ℥j.), or resorcin, carbolic acid, salts of mercury, chrysarobin, pyrogallol. Such agents directly kill the surface epidermis, and do so more quickly than the micro-organisms that may be contained in it (Sabouraud). This sudden killing of the surface cells is followed by rapid exfoliation and formation of a healthier surface beneath. In chronic cases we require to repeat this exfoliative process, it may be several times, before we succeed in dispersing the excess of lymph, and in obtain-

ing a surface of sufficient inertness, cohesion, and stability to resist the natural perturbations of the outside world. In all this it must be borne in mind that it is not a question of killing microbes by germicidal remedies. For, as Sabouraud has shown, the surface tissue killed by the irritant treatment contains the micro-organisms in a living and active state. Our remedies have simply hastened a natural process of expurgation of injured epithelium.

II. *Choice of Remedies.*—From what has been said it will be understood that the choosing of a remedy is not a matter of chance, but one which is regulated by certain definite principles. In selecting a remedy we must know clearly and precisely what it is we have to do. In a word, *what is the stage in the natural process of cure which we desire to anticipate?* It will be found convenient to arrange our remedies in the following classes :—

1. *Passive Agents.* — These include neutral powders, vegetable and mineral, such as oxide of zinc, calamine (artificial and native), stearate of zinc, carbonate of magnesium, carbonate and oxychloride of bismuth, lycopodium, wheat starch, talc, kaolin (cimolite), kieselguhr, chalk. It may be noted here that pure native calamine is very difficult to procure, and in case of doubt it is better to employ the artificially prepared carbonate of zinc coloured to the required tint by the addition of a little carmine. It does not turn black in the presence of sulphur (Brooke). It is decomposed by salicylic acid, a disadvantage not shared by the pure native calamine.

2. *Desiccators and Astringents.*—The above-named powders may also be considered as passive desiccators. Astringents may be regarded as active desiccators inasmuch as they contract the tissues (and blood-vessels), and so actually diminish the volume of fluid in the skin. The best astringents in regard to eczema are silver nitrate, alum, and subacetate of lead. They may be combined with advantage. Thus silver and lead go well together; also alum and subacetate of lead, but in this case decomposition occurs with formation of acetate of aluminium. They must be dissolved in distilled water. The quantity need not exceed 2 or 3 grains to the ounce. A few drops of glycerine and mucilage of acacia may be added when it is desired to protect the surface from friction.

3. *Irritants.* — These may be conveniently subdivided, according to their degree of irritation, into irritants of the first, second, third, and fourth degrees. It must be remembered, of course, that the degree of irritation depends not only on the intrinsic properties of the active agent, but upon its strength. We may roughly classify them as follows :—

(*a*) *Irritants of the first degree.*—Boric acid, borax, ichthyol, etc.

(*b*) *Irritants of the second degree.*—Sulphur, wood, and coal tar, salicylic acid, resorcin, subchloride of mercury, etc., in quantities not exceeding 2 per cent of the basis ointment.

(*c*) *Irritants of the third degree.*—Sulphur, wood, and coal tar, salicylic acid, resorcin, carbolic acid, styrax and Peruvian balsam, iodine, white precipitate, etc., in quantities ranging from 2 to 10 per cent.

(*d*) *Irritants of the fourth degree.* — Chrysarobin, pyrogallol, creasote, corrosive sublimate, red iodide of mercury, and the agents of class (*c*) in quantities exceeding 10 per cent.

III. *Modes of Application.*—Close attention must be paid to technical details in order to secure success in the management of eczema. The action of a drug applied to the skin varies not only according to its intrinsic quality, but according to the way in which we apply it.

1. *The Continuous Method.* — Water and lotions may be applied continuously by means of compresses, with or without the protective covering of oiled silk or gutta-percha. The method is excellent in its results, but makes a greater demand on the time and attention of the patient than some other forms to be mentioned. It is almost indispensable in the treatment of erythematous eczema of the ears. Ointments applied on flannel and butter-cloth are naturally more efficacious than when applied intermittently. *Water* itself is a valuable remedial agent in the hands of those who know how to handle it. The *hot bath* (98°-100° F.) softens the surface of the skin, and also facilitates the escape of the pent-up plasma. At the same time it relaxes the dermal capillaries over the entire surface of the body, and so tends to equalise the cutaneous circulation. Its soothing effect is very great, and it is often chiefly for this reason that we emply the hot bath. It is specially indicated where the inflammation is of a nervous character, as in œdematous eczema of the face, which is not infrequently associated with mental and general nervous excitement. The soothing influence exerted by the hot water on the nerves of the skin is communicated to the cerebral and mental cortex, and to the lower nerve-centres, with much benefit to the inflamed area. The sedative effect of the hot bath is increased by the addition of *bran* or *oatmeal.* About two or three pounds should be sewed up in muslin and boiled for ten minutes in a saucepanful of water; the bag and the water in which it has been boiled should then be added to the bath. The detergent action of water is increased by the addition of *alkalies or alkaline salts,* such as soda or bicarbonate of potash. Enough of the salt should be added as suffices to impart a slight alkaline taste to the water (30 gallons). In some instances *ink* baths are given on account of the astringent action of the iron salt, but their practical value does not appear to be very great. The addition of sulphur is often of value. Instead of the crude

sulphuret of potash, finely-divided nascent sulphur may be formed in the bath by the use of "sulphaqua"[1] charges. They are much to be preferred to the old form of sulphur bath.

Plaster muslins. — Unna has introduced a large number of these, which are made by Beiersdorf of Hamburg by incorporating different medicaments in a basis of gutta-percha and lanolin spread upon muslin. They are an elegant mode of application, and, on account of their impervious nature, have a more penetrating action than ointments.

Salve muslins. — These are medicated ointments spread on one or both sides of muslin, but without the addition of gutta-percha.

Paraplasts are similar to the plaster muslins, but are made with caoutchouc instead of gutta-percha, and are thinner and somewhat more flexible.

Varnishes are fluid applications which, on drying, leave behind a hard film. In theory one might expect them to be a very useful mode of application, but in my experience they have proved of very limited value. Film-forming applications are indicated where it is desirable to prevent friction. I have not met with a varnish which can resist the heat and friction of the groin and perineal groove, where, theoretically, a varnish is often indicated. The collodion and albuminous varnishes are of most value on account of the pressure which they exert on drying. The varnishes employed at present are (1) etherial, or benzine, solutions of india-rubber, nitro-cellulose, gutta-percha; (2) spirit solutions of resins, wax, fats, oleates, and soaps; (3) watery solutions of gum, dextrin, albumin, gelatine, thiol, and ichthyol. Elliot has advocated bassorin as a basis for varnishes. "Gelanthum" is made according to Unna's formula of tragacanth, gelatine, and glycerine. It is intermediate between an ointment and a varnish, but it does not keep well.

Zinc gelatine.—This is a preparation made of glycerine, water, gelatine, and zinc oxide in varying proportions, according as a stiff or a soft gelatine is required. It is in many cases of great value. The gelatine is melted in a gallipot, and, when sufficiently cool, painted on the affected part until a uniform covering has been obtained. This is allowed to set, but, before it is dry, should be dabbled over with absorbent cotton-wool, the fibres of which adhere to the sticky surface, forming a firm dry covering. One of Unna's formulas for zinc gelatine is :—

Zinc oxide, 30·0; gelatine, 30·0; glycerine, 50·0; water, 90·0.

2. *The Intermittent Method.*—Where circumstances do not permit of continuous treatment, we fall back on the use of ointments and lotions. The requisites of a good ointment are sticking power, a not too low melting-point, and that it does not neutralise or be incompatible with the active properties of the included medicaments. Whenever it is desirable not to increase the heat of the part affected, the ointment should be rendered *porous* by the addition of some neutral powder, such as zinc oxide, calamine, or starch. Lassar's paste is constructed on this principle, and has the following formula :—

Zinci oxidi ʒj., amyli ʒj., vaselini ʒij. Fiat ung.

The addition of water, or lime water, to a fatty ointment is often advantageous, on account of the cooling sensation imparted to the skin. The unguentum aquæ rosæ (B.P.) is a good specimen of a cooling ointment; its thinness is often a disadvantage, but this can be mitigated by the addition of ʒj. or more of zinc oxide to each ounce of cold cream.

The question often arises in respect of *the choice between an ointment and lotion* in any particular case. The question is of real importance, as success or failure may turn on our decision. It must always be borne in mind that the action of fat (*per se*), when applied repeatedly to the skin, is to relax the surface tissues, to lessen the pressure on the capillary vessels, and to diminish the loss of water by perspiration. These effects are most apparent on the scrotum, which, under the influence of the fatty inunction, becomes soft and flabby. Therefore, whenever it is desirable to keep the skin well braced up, avoid ointments and choose lotions. This is specially indicated in cases of eczema of the scrotum (see page 41). For the same reason ointments, as a general rule, should be avoided in all cases in which we have to deal with a strong hypostatic tendency, as in eczema rubrum, or varicose eczema, below the knees. Ointments, on the other hand, should be employed in preference to lotions in cases where hardness of tissues is present, where it is obviously of advantage to relax the surface tissues, as in horny eczema of the palms and soles. When in doubt as to the choice of an ointment or lotion, prescribe a porous ointment or paste, which may be regarded, therapeutically, as intermediate between a lotion and an ointment.

Constitutional Treatment.—In the treatment of the mycosiform or the accidental eczemas we need hardly consider the question of internal treatment. But in the management of the intrinsic eczemas, in which the surface tissues play the leading part, we must never forget that we are dealing with an individual who suffers not only from an actual injury to the skin, but also from an increased liability to further injury. It is the nerves that we have to bear specially in mind, since they are mainly responsible for increasing the liability to injury. Whatever remedy, or influence, is able to

[1] These presumably consist of the acid sulphate of soda and the dried hyposulphite of soda, wrapped up separately, the former in lead paper, the latter in parchment. When dissolved in separate basins of water and then mixed, chemical decomposition occurs with the formation of nascent sulphur.

strengthen the vaso-motor control, to regulate nerve action, to reduce the tendency to ganglionic explosions, is able also to render service in the cure of eczema. There is no specific drug nor any single line of treatment to be followed by blind routine. Most internal remedies, which custom has generally approved, often fail because they are given aimlessly. Ergot, strychnine, quinine, ichthyol have done good service in properly chosen cases, but the list of their failures is not a small one.

Climate and Atmospheric Influences.—Patients suffering from irritable nervous eczema should be careful to avoid maritime climates and localities exposed to brine-laden winds. But the more phlegmatic forms of eczema are often benefited by sea or ocean air. We may take it as a rule that inland localities are preferable, provided they are neither damp nor relaxing. It is the excellent situation of Harrogate which has helped to establish its high reputation as a health resort for eczema patients. We must never forget that the therapeutic value of "change of air" is partly to be attributed to what is neither air nor climate, but the fact that the patient leaves behind him his worries and fretful circumstances. He is not likely to be much benefited unless he can go away with an easy mind. And this must be considered before giving him advice on the matter.

Diet.—The importance of diet in eczema is commonly exaggerated. As a rule more harm than good results from meddlesome interference with the natural instincts for food. Experience has taught the writer that in the vast majority of cases of eczema it is unnecessary to specially diet the patient, provided of course there are no complications arising from over-indulgence or injudicious habits of eating and drinking. The exceptions to this statement are the neurotic and gouty cases, in which a purely milk diet for a few days during and subsequent to the climax of the attack is of decided benefit. The gouty man should be encouraged to partake liberally of vegetables, especially spinach and Brussels sprouts. As regards wines and spirits, all alcohol should be interdicted during the attack. For the gouty the safest drink after recovery is probably whisky well diluted with water or salutaris. A little sound claret or hock is permissible.

Part II. Clinical Types of Eczema—Their Diagnosis and Treatment

It is impossible to portray all the symptoms of eczema in its several forms within the limits of a single picture. But if the designs are almost innumerable, they are nevertheless all drawn by the exudate and coloured by the blood. Out of these two elements are constructed an infinite number of pictures, according as we have more or less blood fluxion and more or less exudation. Thus an abundant serous exudate approaching the surface against resistance produces, with the hyperæmia, the picture of vesicular eczema. If there is little or no resistance to the escape, the plasma exudes in a general ooze, as in eczema rubrum. The drying and coagulation of the plasma on the surface produces scabs. The casting off of the injured œdematous epithelium in thin membranous or bran-like portions produces scaling, and, if deeper portions of the epithelium are thrown off, scabs. If the exudate is so moderate in quantity as not to be visible on the surface, it results in a circular, circumscribed "dry" patch, more or less coloured according to the degree of blood fluxion. The picture is further modified according to the stage of repair, the aspect being scaly, scabby, and rough before separation has occurred. On the other hand, when the new horny layer is just complete, and the old one has been thrown off, the aspect is that of a glazed, polished surface, often of a livid colour, owing to the extreme slowness of the capillary flow and the relatively excessive quantity of suboxidised blood. Since no two cases of eczema are likely to be in precisely the same stage of evolution when seen by the medical adviser, and as every square inch of eczematous skin may include different stages of the process of expurgation at the same time, it is quite obvious that the several types of eczema cannot be made to depend on its lesions.

I. *General Character of the Accidental Eczemas.* —(See p. 26.) There is nothing absolutely distinctive in the characters of the accidental eczemas, and in some cases it is hard to distinguish them from the true intrinsic eczemas. Usually, however, the following points determine the diagnosis :—(1) history, suddenness of onset, rapidity of course; (2) the sharp limitation of the eruption to the area of external irritation; (3) often (but not necessarily) the uniformity of the lesions; (4) the presence of excessive deep-seated œdema; (5) intensity of the process with excessive exudation.

II. *General Character of the Intrinsic Eczemas.*

I. *Acute Eczema.*—The words "acute" and "chronic" are employed in this article merely to denote the pressure at which the exudate enters and passes through the epithelium, and are not intended to indicate the duration of the course. An acute or high-pressure eczema is preceded by a sensation of burning and itching in some part of the cutaneous surface, where a diffused or punctate redness soon becomes visible. The spot becomes hotter and more irritable, and there is an irresistible impulse tô rub or scratch it. The usual smoothness and firmness of the surface disappears; it becomes softer and moister, also puffy and swollen; vesicles appear, and, slowly or quickly pushing out the cuticle, cause it to break; the plasma exudes on to the free surface, and dries to a scab. Around the primary focus the eruption

is seen to be in various stages: first, vesicles, broken and unbroken; beyond this, bright red papules; and, still farther away from the focus of disturbance, hyperæmic spots (maculæ). On the outermost margin of the disturbed area the epithelium may appear more translucent than normal, and here and there a streak of a capillary can be seen. Sooner or later fusion occurs of all the several spongy spots where the irritation has been greatest, and a uniformly reddened patch is formed, the surface of which is rough and scaly, with dry, or drying, scales adhering, and here and there a glazed, smooth spot. Usually there are marks of excoriation where the finger-nails have been. The margin of the patch slopes gradually down into the pale skin, and is surrounded, usually, by many isolated red papules and spongy points. In acute eczema with a high degree of infectivity (non-bacterial) the inflammation spreads fairly symmetrically on both sides of the body, also upwards and downwards. Thus eczema starting behind one knee will soon appear behind the other, and will tend to spread up the thigh and down the leg. Acute eczema of the genitals tends to spread upwards on to the abdomen, and downwards on to the thighs. Acute eczema of the scalp tends to descend over the forehead, ears, and face to the neck and shoulders. Acute eczema in any part of the cutaneous surface is apt to be followed by the development of eczema in any region, the surface of which has been weakened by injurious habits of life, or by previous attacks of eczema. The practical bearing of this is obviously that in all cases of acute local eczema we should examine carefully from time to time all regions exposed to friction, heat, or the macerating action of perspiration. In rare cases eczema becomes almost universal. It is remarkable how little constitutional disturbance accompanies even the worst forms of disease. In cases where its development is rapid and extensive there may be considerable nervous excitement, which may show itself in trembling of the limbs and in mental irritability. This is specially marked in alcoholic patients, and in persons of neurotic temperament. But there is no fever and no decided feeling of unwellness.

2. *Chronic or Low-Pressure Eczema.*—Most dermatologists are agreed that eczema is essentially a chronic process subject to acute exacerbations. It is always slow and insidious in its development, although the actual attack may appear acute and sudden. When the high pressure period is passed it sinks into the indolent, stagnatory condition, which is the most marked of the characters of eczema. There are many clinical varieties of chronic eczema. The intrinsic (non-parasitic) forms, with which we are alone here concerned, are the papular, the indurated, the cornified, and the follicular.

(1) *The Papular Form.*—Eczema may begin, continue, and end as a papular eruption. The papules are conical, round-topped elevations of a brighter or darker red colour developing in close proximity, and varying in size from a small to a large pin's head. The eruption was formerly known as *lichen simplex.* In certain regions of the body, *e.g.* the shoulders, they may acquire a smooth, flat-topped aspect, to which Brooke has drawn special attention. Eczematous papules are, as a rule, indolent and persistent. Where overcrowded they run together to form a dense infiltrated patch. The itching is severe, in some cases intolerable and maddening, and leads to violent scratching. Hence in these cases the papules are usually crowned with a minute blood scab. After prolonged or violent scratching the eruption may acquire in some regions quite a peculiar lichenoid aspect, well described by Brocq of Paris, in which there is great induration of the skin, with deepening and intensification of the surface lines and furrows.

(2) *Indurated Form.*—Eczema does not necessarily involve the derma; for it is essentially an epithelial-neuro-capillary affair. When the process involves the derma it results in a sclerosis or fibrosis. In these cases an additional factor, the nature of which is still uncertain, comes into operation. Dermal induration does not altogether depend on time. Some persons have a stronger tendency towards sclerosis than others. The difference seems to depend on inherited trophic peculiarity. In certain cases eczema passes rapidly from the acute condition into the indurated form; while in others it may persist for months without showing any tendency to pass into sclerosis.

(3) *Cornified Form.*—In this there is a hypertrophy of the horny layer; it is commonly met with in the palms and soles (see under Regional Varieties for further description).

(4) *Follicular Form.*—This was first described as a special form of the disease by Malcolm Morris. The inflammation begins in the hair follicles. The eruption consists of angry-looking red papules surrounded by congested capillaries. Where overcrowded the papules fuse together to form red patches dotted with inflamed follicles. While the inflammatory process spreads at the margin, resolution tends to set in at the original focus of the disease. The itching is often intense. The patches are generally multiple and scattered over the exterior surface of the arms and legs. This form is placed provisionally in the class of intrinsic eczemas, but it is not improbable that future research may warrant its removal to the group of mycosiform eczemas.

III. *General Character of the Mycosiform Eczemas* (*Seborrhœic Eczemas, etc.*).—Some authorities are inclined to transfer these eczematous forms altogether from the class of true eczemas to the group of seborrhœides. They

are fairly entitled, however, to be included among the eczemas, as they manifest all the pathological elements of genuine eczema, namely, exudation into the epithelium with expurgation of the injured tissue and new epithelial growth. So little is certainly known of the bacteriology of these forms, or of the relationship which exists between the inflammatory manifestation and the accompanying seborrhœa, that the present writer prefers to speak of them collectively as mycosiform eczemas. There can be little doubt that the group contains several varieties which both clinically and bacteriologically are as yet ill defined. According to Unna, to whom we are chiefly indebted for what information we possess, they are all examples of seborrhœic eczema, in which the exudate is remarkably rich in fatty products, derived, in the main, from the sweat glands, the efficient cause of the whole fatty-serous catarrh being the morococcus. This conclusion will probably be modified by future research.

Clinically all the mycosiform eczemas are distinguished by a low-pressure exudate which usually (apart from complications) does not show on the surface, but filters into the epithelium. The vascular paralysis is strictly limited to the area of the injured œdematous epithelium, and, in consequence, the outlines of the efflorescence are circumscribed, figured, or serpiginous. The commonest and most characteristic lesion is a circular hyperæmic disc covered with greasy yellowish scales, which can be easily torn off. These scales may exhibit little bags of fatty matter on their under surface derived from the follicles. This seborrhœic variety of mycosiform eczema usually descends, as Unna has shown, from the scalp, where it begins as a *pityriasis capitis* (common dandruff). It may remain in this condition for months or even years. A certain proportion of these cases (the ratio not yet ascertained) pass into the inflammatory condition. The earliest signs are to be found at the junction of the forehead with the scalp, in the form of dry, red scaly patches with a more or less defined margin. Soon signs of eczema appear behind the ears. The post-aural grooves become reddened and frequently fissured. This condition is usually aggravated by scratching, and it is not rare to meet with an acute weeping eczema involving the ears and temples. Early in the course of the disease we may detect yellowish-red, greasy, circular patches over the sternum. In many cases the inflammation surrounds the neck as a dry red collar, and may attack the bends of the elbows, the genital-groin region, and the bends of the knees. In rare cases the disease may become almost universal. It is surprising how superficial the inflammation is; even long-standing cases may show no signs of dermal induration. This character is so constant that it may be said that if a patch of eczema is notably indurated it is probably not a mycosiform eczema.

The *epidemic eczema*, described by Savill, probably belongs to the class of mycosiform eczema. The cases described by this author occurred for the most part in old, bed-ridden people. The type is mainly that of a dry catarrh (low-pressure eczema) assuming various lesional forms, which in the early stages spread in ringed and serpiginous patches, and in their latest and worst development are indistinguishable from pityriasis rubra. The contagiousness of the disease is proved by the large number of patients who were attacked at the same time in the Paddington and Marylebone Infirmaries in London. The disease is said to have been conveyed to animals. Savill cultivated an organism which was constantly present in the scales and exudate. It was an aerobic diplococcus growing in all soils, and not liquefying gelatine. The description tallies closely with that of Unna's morococcus.

Regional Forms of Eczema [1]

The Scalp.—*Type* 1. *The hair is falling out; the surface is covered with greasy scales, but is not obviously hyperæmic; red scaly spots appear at the junction of the forehead and scalp; the nape may be suffused with a pink colour and perhaps scaly.*

Treatment.—This is a mycosiform variety and intimately associated with seborrhœa. It requires powerful detergent and stimulating treatment. Wash the head for the first week every night with spiritus saponis viridis (Hebra), thoroughly removing the lather with water, and immediately afterwards applying an ointment containing ʒj. of coal or wood-tar to the ℥j. If the tar is objected to, substitute sulphur (ʒj.) and salicylic acid (gr. x.-xx.). When the eruption has been removed the scalp must still be washed once or twice a week, and a more agreeable ointment (salicylic acid, or resorcine in white vaseline) prescribed. Later a lotion may be ordered containing oleum ricini (ʒss.-ʒj.) and salicylic acid (gr. x.-xv.) to ℥j. of rectified spirits.

Type 2. *Similar to type* 1, *but the temples are acutely inflamed and weeping; the ears are red and swollen.*

Treatment.—This variety has the same origin as the preceding one, but is greatly aggravated by the presence of decomposing discharge, and by scratching. No fatty ointment should be applied in this stage, for the obvious reason that the parts are already too relaxed. The desiderata are drying, astringency, and asepsis. Compresses wet with acetate of aluminium (aluminii sulphatis gr. iii., plumbi acetatis gr. i.-ii., aquæ distil. ad ℥j.), or with silver nitrate

[1] Owing to limitations of space it has not been possible to allude to the many excellent and successful forms of treatment suggested by other writers.

(gr. ii. to the ℥j.) in distilled water. When the discharge has ceased the surface will have a red, glazed appearance. This is merely a temporary surface, and will be certainly thrown off. Begin gentle stimulating treatment. Mild sulphur liniment is excellent, such as sulphur gr. x., ol. olivarum, aquæ calcis āā ℥ss. When the surface becomes dry, pale, and rough, treat as in type 1.

Type 3. *The patient is debilitated, perhaps alcoholic; the surface of the scalp is acutely suppurating in places, and boggy and œdematous in others.*

Treatment.—Here astringent lotions or porous ointments are indicated, and not fatty ointments. As soon as the scabs have set and hardened, and the tissues are sufficiently contracted, the head must be washed with the spirit solution of soap, and stimulating ointment treatment begun. The ointments are all the better for being a little porous in these cases, and this is effected by the addition of ʒj. or more of calamine or zinc oxide to the ℥j. If sulphur and salicylic acid in combination fail, pyrogallol (grs. v.-x. to ℥j.) should be tried.

Type 4. *The eczema is associated with a scalp wound.*

Treatment.—Drying and astringent treatment is very important here. Lassar's paste (see p. 33) with 2 per cent salicylic acid is an excellent thing to begin with, and should be continued till the surface is firm and dry, when soft fatty ointments may be employed as recommended in type 1.

Face (smooth parts).—*Type* 1. *The patient is a teething child; the surface of the forehead and cheeks is thin, red, and moist, the cuticle having poor protective qualities.*

Treatment.—The nerve irritation occasioned by the cutting of the teeth is apt to aggravate the eczema. Care must be taken to cut off all sources of reflex irritation, as far as possible. The food must be human or cow's milk, the latter suitably diluted according to the age of the child. Locally the skin must be screened from the air. Unna's zinc ichthyol salve muslin is of great value here. It has, however, very little astringent action. If the parts remain moist in spite of its use, astringent lotions must be applied in compress form. When the surface is dry a slightly stimulating ointment containing sulphur (grs. x.) and tar (ol. rusci ♏x.) should be smeared many times a day over the whole face.

Type 2. *The face of a child is covered with pus scabs, seated on a moist and inflamed skin.*

Treatment.—Purulent eczema is very common in children. Lassar's paste, with 1-2 per cent of salicylic acid, or some similar antiseptic porous ointment, is indicated. When the surface is dry employ more stimulating ointments or lotions. An excellent preparation for final treatment is tar-calamine lotion.

Type 3. *The patient is a highly neurotic adult person, generally of the male sex. The surface is dry, but hot, red, and much swollen, especially about the eyes, so as to prevent their being opened. The irritation is severe, in some cases intolerable, producing great mental excitement. The symptoms are subject to sudden variations, either in the direction of amelioration or of exacerbation.*

Treatment.—This is a peculiar type of eczema, in which there exists an extraordinary sensitiveness of the neuro-capillary apparatus. We know nothing as to the actual determining cause. In the treatment of this type all active local remedies are contra-indicated. Bland creamy ointments are all that are required locally in the early stage. A liniment of olive oil and lime water, with a little zinc oxide or calamine to thicken it, generally answers well. The patient must be confined to one room, and even kept in bed if the symptoms are very severe. The diet during the stormy period must be purely of milk. A 5-grain Plummer's pill may be administered at the outset, or, as Malcolm Morris prefers, the wine of antimony in 5-drop doses repeated half-hourly till the force of the pulse begins to abate. Such depressing remedies are only to be used at the height of the nerve storm. With the subsidence of the symptoms a more liberal diet may be allowed, but no red meat and no alcohol. Before the patient leaves his room, astringent lotions must take the place of the ointment for the purpose of hardening the surface so as to enable it to resist wind and sunshine. This form of eczema indicates nerve fatigue, and is apt to occur when the mental output is greater than the cerebral intake of nutriment. The ordinary diet of the patient should be rich in phosphates. If he is also gouty he should be encouraged to partake of such vegetables as spinach and Brussels sprouts. While nerve tonics are advisable, it must not be forgotten that the best of restoratives are change of air and scene. Dry exhilarating mountain air is much to be preferred to sea-level climates; maritime localities, in fact, are liable to prove injurious.

Type 4. *The patient is a scrofulous child with dry, well-defined patches of eczema situated at the junction of skin and mucous membrane.*

Treatment.—This is the variety which Unna names "*tubercular eczema.*" It tends to develop in skins of a scrofulous type (see p. 28), and is remarkably rebellious to treatment. The most satisfactory method in the writer's experience is by means of the plaster muslins, of which the carbol-mercury and the tar salicylic plasters are to be highly recommended.

Hairy Regions of the Face.—Eczema of those portions of the face covered by the beard and whiskers has to be distinguished from sycosis, tinea barbæ, and impetigo. Sycosis is essentially a follicular and peri-follicular inflammation of

coccogenous, bacillary, or trichophytic origin, and is usually a purulent form of inflammation. It tends in the chronic cases to perifollicular induration, thus forming what are clinically known as "nodules." The certain diagnosis of dry eczema from dry superficial tinea of the beard can only be made by the aid of the microscope. Impetigo is distinguished from purulent eczema of the face by the fact that the suppuration forms practically the whole of the impetigo process, while in eczema it is only a part of a general serous inflammation. Consequently in impetigo the inflammatory symptoms do not extend beyond the circumference of the pus scabs, which thus convey to the eye "the appearance of having been gummed on." In eczema, on the contrary, the scabs appear to be adhering to a red catarrhal surface.

Type 5. *Red, dry, scaly patches are scattered through the beard and whiskers; they are quite superficial and do not affect the follicles; they are associated with pityriasis capitis.*

Treatment.—This is a mycosiform (seborrhœic) eczema. There is no need to shave the beard or whiskers. A thin paraffin ointment containing ʒj. or more of oleum rusci with ʒj. of precipitated sulphur is to be well rubbed in with the fingers. If preferred, a spray of liquid paraffin containing ʒj. of oleum rusci to the ℥j. may be used three or four times a day.

Type 6. *The skin in the region of the beard and whiskers is of a dull red colour, infiltrated and thickened to a moderate extent; a few follicles are suppurating. The disease is of many months' or years' duration.*

Treatment.—This variety is known as sycosiform eczema from its resemblance to sycosis. The inflammation, however, is not essentially follicular, as in the latter disease. In the treatment of these chronic cases care must be taken not to over-stimulate the weakened and damaged skin by irritant remedies, which have the injurious effect of setting up fresh suppuration. If possible the hair must be shaved, or at least clipped close with the scissors. It is well to begin with a liniment such as the following:—

Ol. olivarum,	
Aquæ calcis, āā	℥iij.
Zinci oxidi	ʒiv.
Sulph. precip.	ʒiij.
Hydrarg. subchlor. . . .	ʒss.

This must be dabbed or painted over the affected parts many times in the day. It must be continued until all suppuration has ceased, and the hyperæmia and tension have been reduced to about half their previous extent. After this the 5 per cent oleate of mercury and Lassar's paste in equal parts, with 1 per cent of boric acid or salicylic acid, should be gently rubbed in three times a day.

Type 7. *The upper lip covered with the moustache is the seat of a patch of chronic inflammation having a somewhat sharply defined margin; it may, or may not, be scattered over with superficial pustules, but there is no follicular induration. This variety is met with in old alcoholics.*

Treatment.—This form is extremely rebellious, and may persist for months or even years. In the experience of the writer the only remedy which has any influence over it is mercury. The ointment recommended by Brooke for lupus does well here. The formula is—

Hydrarg. oleatis, 5 per cent .	ʒiv.
Pastæ Lassar . . .	ʒiv.
Acidi salicylici . . .	gr. x.
Ichthyolis	gr. x.

This must be gently rubbed into the affected parts morning, afternoon, and evening. These patients require strict supervision on account of their loose, intemperate habits.

Lips.—*Type* 1. *The lips are œdematous, much swollen and immobile; also covered with vesico-pustules and crusts; the condition spreads on to the parts around the mouth.*—Begin treatment with an antiseptic porous ointment (Lassar's paste with 2 per cent salicylic acid), or with zinc-ichthyol salve muslin. When the surface is clean apply mild astringent lotions containing silver nitrate (gr. ii.), or blackwash and olive oil mixed in equal parts.

Type 2. *The lips, especially the lower one, are much swollen, dry, and red; the cuticle covering the lips is peeling, cracked, and even deeply fissured.*—This is an extremely rebellious affection. In the writer's experience the only treatment which has given good results is that suggested by Kaposi. It consists in painting the lips with a 30 per cent solution of caustic potash in water, and immediately after neutralising the alkali with vinegar or dilute acetic acid. For a few days after this operation the swelling of the lips is increased, but this is followed by a marked lessening of the inflammatory symptoms and reduction in the size of the lips. Unna's ichthyol varnish is a valuable addition to Kaposi's treatment. When the œdema following the application of the alkali has subsided, the varnish should be painted on the lips every night. It is not suitable for application by day unless the patient is confined to the house.

Eyelids.—These are specially prone to be affected in scrofulous children with chronic inflammation, in which the exudate is apt to be purulent, and is peculiarly rich in fatty products from the Meibomian glands. Mercurial ointments hold unquestionably the first place in the treatment of this affection. The unguentum hydrargyri acidi nitrosi dil. (B.P.) in the proportion of ʒj.-ʒiss. to ℥j. of lard is usually sufficient to cure the disease. The application of a solution of silver nitrate in distilled water (gr. x.-xv. to the ℥j.) may be necessary in very rebellious cases.

Ears. — *Type* 1. *Acute inflammation with great swelling and serous discharge.*—Eczema of the ears is liable to sudden acute exacerbations, in which the parts become enormously swollen, red, hot, and weeping. In this condition ointments must never be applied to the ears. The continuous application of muslin compresses wet with a solution of silver nitrate (gr. ii. to the ℥j.), or of alum and acetate of lead, has the desired effect of reducing the swelling, of lessening the hyperæmia, and of arresting the discharge. After a few days, when the ears have nearly assumed their normal size, gentle stimulation may be employed in the form of a liniment composed of equal parts of lime water and olive oil with a little sulphur (gr. v.) and subchloride of mercury (gr. v.). Eczema of the ears is often associated with inflammatory seborrhœa of the meatus. It is essential to a permanent cure that this should be treated. If the surface of the meatus is soft and œdematous an astringent lotion is indicated, and its use may be followed by a sulphur-resorcine ointment smeared on a plug of cotton-wool, and inserted in the meatus. This should be renewed every six hours. Accumulation of wax or of discharge should be syringed away, after softening, with a solution of borax in water. If there is daily discharge, especially if of fœtid odour or streaked with blood, the middle ear is probably involved. Eczema of the ears is usually accompanied by seborrhœa of the scalp, and this must be treated according to the directions given above (see Scalp, Type 1).

Type 2. *The parts behind the ears are red and fissured, and covered with greasy scales and scabs.* —This is generally a part of mycosiform (seborrhœic) eczema of the scalp and other parts of the body. Ointments are borne well. Porous ointments or pastes are better than simple fatty ones. If the surface is moist, begin with Lassar's paste with 1 or 2 per cent salicylic acid. When the surface is dry and scaly, a tar-zinc-sulphur ointment answers admirably. The proportion of sulphur and tar must vary according to the degree of stimulation required.

Type 3. In scrofulous children a very rebellious type of eczema is met with behind the ears; the parts are red, rough, scaly, and indurated, the affected area being limited by a sharp margin. It may resist all the usually successful remedies. Such cases may with advantage be treated with Unna's mercury-carbolic acid plaster muslin (No. 88). One application each day is sufficient.

Neck.—In well-marked cases of seborrhœa of the scalp the nape seldom fails to show signs of hyperæmia. It may be nothing more than a pink coloration, but in many cases it becomes the seat of a very obstinate chronic eczema. Strong stimulating remedies are required here. Coal-tar is to be especially recommended, and may with advantage be dissolved in spirits of wine with the addition of a little ether. The erythematous collar which forms around the neck is commonly met with in the mycosiform eczemas which descend from the scalp; it is important, therefore, in those cases always to examine the scalp. A lichenoid form of eczema develops in some cases on the shoulders of men. Very powerful local stimulants are necessary to cure it; and care must be taken to remove the pressure of the braces.

Trunk.—This is usually the seat of dry mycosiform eczemas.

Type 1. *The chest and back are covered with a dry, patchy eczema, approaching clinically to the type of psoriasis.*—In seeking to distinguish this psoriatiform type of mycosiform eczema from typical psoriasis it is necessary to bear in mind that the two diseases may occur simultaneously in the same person, and that in some cases the inflammation may partake partly of the character of psoriasis and partly of that of a dry seborrhœic eczema. In psoriasis the silvery appearance of the scales seated on a dry hyperæmic base, and their main distribution on the tips of the elbows, the fronts of the knees, the extensor surfaces and scalp, together with the chronicity and periodicity of the eruption, serve as a rule to distinguish the disease from eczema. If any further doubt remains the examination of the scalp usually settles the difficulty; for psoriasis develops in this situation in island-like patches, and does not lead to the death of the hair as seborrhœic eczema capitis almost invariably does. The treatment is practically the same as for psoriasis. Sulphur and tar must be pushed boldly (ʒj. to ʒij. to the ℥j.); and if these are too weak, chrysarobin (gr. xx. to xxx.) may be relied upon to remove the eruption. This form of eczema is not incompatible with the daily bath. There is no need to specially diet the patient. Internal treatment is not required so far as concerns the eczema.

Type 2. *The surface is very red, and actually moist in certain places; other parts are occupied by numerous bright red papules; there is great vaso-motor irritability, and the whole inflammatory process is spreading.*—If there is no renal or cardiac disease, and no arterial degeneration, and the patient is not too debilitated, a full-length warm bran-bath may be given. The temperature should not exceed 100°. Circumstances must determine the duration of the bath; it may vary from quarter of an hour to an hour or even longer. A single bath may suffice; for all that is required of this water treatment is to relax the tissues, to aid the removal of the discharge, and to equalise the cutaneous circulation. If this is not attained the first day, the bath must be repeated the following night. After the bath the body must be carefully dried, and at once anointed with a cooling and porous ointment. Cold cream

with ʒij. of zinc oxide to the ℥j., and 1 per cent of boric acid, or 2 per cent of salicylic acid, is a suitable form of treatment. A cheaper ointment is benzoated lard ℥j., calamine ʒj., and salicylic acid gr. x. When the inflammatory symptoms have subsided, and the surface has become rough and scaly, tar and sulphur in gradually increasing strength are indicated. In females with eczema under the breast it is absolutely essential to separate the parts so as to prevent contact; zinc ichthyol salve muslin is useful for this purpose. When this cannot be obtained, a porous ointment, such as Lassar's paste (see p. 33), with 2 per cent salicylic acid, spread over muslin, must be inserted under the breasts.

Axillæ.—Eczema in these regions is apt to be modified by heat, abundant secretion, and constant friction. During the acute stage zinc-calamine lotion with 1 per cent boric acid, or Lassar's paste with 2 per cent salicylic acid, is safe treatment. The subsidence of the symptoms is always to be followed by the more stimulating application of tar, sulphur, or subchloride of mercury.

Arms.—There are only two types which require special description.

Type 1. *The eruption consists of spongy points closely clustered together in patches far apart from each other. The skin is thin, and the neuro-capillary apparatus highly sensitive. Very slight irritation causes a thin, clear serum to ooze out from the spongy points; some of these are temporarily closed with a minute scab not larger than a pin's head of medium size. The patients are in poor health, and frequently are the victims of nervous exhaustion.*—This is a peculiar type of eczema which is to be distinguished from the common varieties by the fact that the degree of inflammation is not proportionate to the amount of epithelial injury, but is determined by the morbid irritability of the neuro-capillary system of the skin. There is a conspicuous loss of vaso-motor control. No form of eczema is more troublesome to the patient or to the physician. The irritation is often intense. Very slight stimulation will cause the discharge to break out afresh. The ordinary remedial ointments only cause fresh serous exudation. The only local treatment required is to cut off all sources of irritation (air, light, friction of clothes, finger-nails), and this is best attained by astringent or alkaline lotions applied in compress form with an over-covering of oiled silk. When the patches have lost their spongy aspect, the compresses may be exchanged for zinc-ichthyol salve muslin, or calamine lotion with a few minims of liquor picis carbonis. Nerve tonics such as quinine, strychnine, phosphoric acid, are indicated, but they will do no good unless we can set the patient free from the circumstances which worry and fatigue his brain and mind. Thus change to bracing hill-side air is often of more value than medicine.

Type 2. *The bends of the elbows are red, dry, and indurated; the natural lines and furrows of the surface are exaggerated.*—Eczema is very common in the bends of the elbows and is apt to become chronic, and to linger there after it has disappeared elsewhere. Here, as also behind the knees, eczema is specially prone to involve the derma, which becomes indurated and sclerosed. From its anatomical position it is subject to disturbances arising from pressure on nerves (prolonged flexion), and the constant movement of parts. Eczema in these regions must always be treated by the continuous method (see p. 32), and usually by ointments in preference to lotions. To remove induration we may require strong tar-sulphur-salicylic ointments. If these fail we must employ chrysarobin.

Hands.—The hands, with the face, are the most nervous regions of the cutaneous surface. Bouts of worry and nerve-exhaustion are specially apt to be followed by the development of eczema in these parts in persons with defective vaso-motor control.

Type 1. *The eruption consists of patches of spongy points in a thin, over-sensitive skin.*—This is the same as type 1 described above under Arms.

Type 2. *The eruption consists of superficial vesicles forming slowly and dispersing slowly, each being isolated, distinct, and well formed; fresh crops of vesicles arise at intervals until the hand on both sides is fairly studded with vesicles; when they are crowded together, œdematous red patches are formed, but in the early stages there is very little hyperæmia. The burning and itching are severe.*—This variety is slow and tedious in its course, owing to the local anatomical obstacles to the dispersion of the plasma, and the throwing-off of the injured epithelium. If the hand is studded with vesicles, and the irritation is severe, the whole of the surface-skin must be removed before a permanent cure can be obtained; and remedies which are fitted to do this, without increasing the irritation, are clearly indicated. The shortest road to this end is complete immersion of the hands in alkaline warm water. When this is not convenient alkaline water compresses may be used. When the cuticle is sufficiently macerated, and the exudate dispersed, the water treatment must be stopped. At this stage the hand looks worse, owing to the predominance of the hyperæmia and the fusion of the lesions into each other. In reality, however, the conditions for healing are much more favourable. The parts must now be covered with a keratoplastic porous ointment. One of the safest of the keratoplastic agents for general recommendation is salicylic acid [1] in proportions not exceeding 2 per cent of the ointment basis, which may be

[1] In stronger proportions salicylic acid displays an action the reverse of keratoplastic, namely keratolytic.

benzoated lard with 12 per cent calamine or zinc oxide. Under this salve the surface soon becomes reformed and hardens, and then requires the addition of a little tar to the ointment. Treatment must be continued long after the surface has become fairly pale and intact, since vaso-motor weakness and softness of the surface remain for several months. Tar-calamine lotion is to be recommended in this final stage.

Type 3. *The palms and fingers are the seat of a rough, corneous, thickened state of the epidermis; one or more of the lines of flexion are fissured and exude plasma; in some cases the eczema involves the cutis, producing more or less induration. The disease is usually symmetrical.*—Only two other conditions are likely to be confounded with this form of eczema, namely, psoriasis and late symmetrical syphilides. As regards psoriasis, a knowledge of the differential diagnosis is of no importance so far as concerns treatment, but it is useful for purposes of prognosis. If the condition of the palms is part of a dry cutaneous catarrh which is worse in autumn and spring, if it has lasted off and on for years, and tends to involve specially the tips of the elbows and the fronts of the knees, the right diagnosis is psoriasis. The psoriatic inflammation of the palm is a dry process which produces red discs surrounded by a free collar of ragged epidermis, imparting to the lesion a worm-eaten appearance. To distinguish from syphilides note the history and course of the disease. If there is no appearance of a serous exudation endeavouring to escape through the thickened stratified epithelium, if there are no moist or dry fissures, if the lesion is conspicuously circumscribed and surrounded by pale skin, and at the same time passes deeply into the cutis with marked infiltration, the diagnosis is in favour of syphilis. A point of prime importance, which Mauriac of Paris has pointed out, is that a genuine syphilide of the palm may in course of time lose all its syphilitic taint, and degenerate into a simple eczema, which is unaffected by mercury or iodide of potassium, but yields completely to suitable local treatment.

Treatment.—The natural indication is the removal of the injured horny tissues, and this is best effected by covering the parts with salicylic plaster muslin containing from 5 to 10 per cent of salicylic acid. Fresh plaster must be applied once daily, and continued till the horny tissue is removed, when zinc oxide or tar plaster may be substituted for the former, and continued till the surface is once more reformed. A stimulating ointment should now take the place of the plaster, such, for example, as salicylic acid (2 per cent), oleum rusci (2 to 5 per cent), zinc oxide (10 per cent in lard or vaseline).

Cheiropompholyx.—It is still a question whether the disease known as dysidrosis or cheiropompholyx should be included among the eczemas. The vesicles of cheiropompholyx are more deeply seated at their origin than those of eczema, and bear a superficial resemblance to boiled sago grains. The most prominent feature is the intense irritation which accompanies their development. The disease is symmetrical, and may attack the feet at the same time. It occurs most commonly in young neurotic women, and is apt to relapse after apparent cure. A mistake in diagnosis is of no importance so far as concerns treatment.

Genital Region.—*Scrotum.*—When eczema thoroughly attacks the genital region it is not easily cured, for it is a region specially liable to injury, to unrest, and to disturbances of circulation, owing to the numerous flexures, its dependent position, and to the fact that it is one of the hottest and most glandular parts of the cutaneous surface. The worse forms of scrotal eczema occur in persons whose muscular scrotal fibres are completely relaxed. A flabby scrotum in a man over fifty years of age, and addicted to the pleasures of the table, will retain more or less of its eczema indefinitely. Owing to the relaxed condition of the fibrous meshwork of the scrotum, the exudation extends laterally and deeply, so that, in acute cases, the organ may be enormously swollen. No form of eczema is more harassing than that affecting the genital region. It commonly spreads to the groins, reddening and cracking the skin at the bottom of the grooves, and may involve the penis, or spread over the abdomen and down the thighs. The anus in long-standing cases is usually involved.

Type 1. *The scrotum is relaxed, swollen, and red, and covered with foul-smelling secretion.*—Treatment may be started by a single warm bran, boracic bath of ten minutes' duration to remove secretion and putrid products; and this should be followed by the application of compresses of mild astringent lotions (silver nitrate, alum, acetate of lead), to be kept in position by means of a pair of white cotton bathing-drawers. The astringent treatment must be continued till the scrotum is well braced up. Later the addition of tar solution to the lotion is to be recommended. Ointments should be avoided.

Type 2. *The scrotum is flabby and slightly swollen; dry with no obvious secretion, but hyperæmic, hot, and probably scaly. The groins are red and fissured.*—Here there is no need for the bath. Astringent lotions should be used as in type 1. Ointments must be avoided. Bathing-drawers must be worn constantly, and the lotion applied on soft flannel or butter-cloth, a separate piece being placed in each groin, and another over the scrotum.

Anus.—Two forms of the inflamed anus are commonly met with—(1) the leathery anus, and (2) the pink anus; both require different

treatment. The irritation which accompanies anal eczema is in many cases of a very distressing nature, depriving the patient of sleep and appetite. It may be deeply seated, passing into the rectum. The condition is usually associated with hæmorrhoids, that is, with local disturbances of the circulation. The inflammation usually passes up the perineal groove, leading to fissuring of the epidermis.

Type 1. *The Leathery Anus.*—Here the parts are hypertrophied and of a dull red colour, in some cases dark brown owing to the increased pigmentation. Zinc gelatine (see p. 33) is well borne in these cases, and usually gives great relief, and may effect a satisfactory cure. Examine the anus internally. Hæmorrhoids and fissures must be treated on surgical principles.

Type 2. *The Pink Anus.*—This is more troublesome to treat than the leathery anus. The parts around the orifice present a bright red appearance, are very soft and tender, and are much disposed to fissure between the anal folds. The soft œdematous condition passes directly into the rectum. The itching is very persistent and is always worse at night. This variety is commonly met with in persons who are prone to rheumatism, or who suffer from some of the atypical symptoms of gout. The writer regards it as an indication of local muscular and capillary relaxation. The pink anus does not usually bear well the zinc gelatine, which fails to adhere to the hot, damp surface. A pad of zinc-ichthyol salve muslin should be inserted between the buttocks, and renewed morning and evening. All sources of rectal irritation must be carefully removed, as, for example, hard stools which necessitate straining. It is not advisable to purge unless there is evidence of intestinal irritation. A little cascara or compound liquorice powder is usually sufficient. The parts should be cleansed with a little boric lotion after stool, and the salve muslin reapplied. The deep-seated irritation may be relieved by a suppository containing a little hamamelin and opium as recommended by Malcolm Morris. Enemata are not advisable, as they usually distress the patient. Diet must be considered. High living is injurious, and all highly spiced and complex articles of food must be omitted from the patient's dietary. Malted alcoholic stimulants are more likely to disagree than good sound clarets or hocks, but the patient cannot be too careful in regard both to the quality and quantity of his stimulants.

Penis.—No special description is needed, as all that has been said regarding the scrotum applies also to the penis. In some cases the prepuce is affected on its inner surface along with the glans. Scrupulous cleanliness with the use of an astringent lotion is all that is necessary to effect a cure.

Vulva.—The treatment of eczema of the vulva is similar to that of the scrotum. Lotions are to be used in preference to ointments.

Groin.—It is important not to confound the so-called *eczema marginatum* with simple eczema of the groin. The former is a trichophytosis which, not seldom, is marked by secondary inflammatory symptoms due to friction and the irritation of decomposed secretions. Whenever the margin of an inflammatory patch in the groin has a sharp serpiginous outline, the probability of a parasitic origin is great, and the microscope must be used to settle the diagnosis. The treatment of parasitic inflammation of the groin must be strong and energetic; most failures are due to the practitioner attempting too little. On the other hand, simple eczema of the groin requires soothing astringent treatment, and in the later stages slightly stimulating remedies. The chief trouble is the constant tendency to the formation of fissures. Silver, lead, or alum lotions, with or without the addition of tar, are the safest remedies.

Legs.—*Type* 1. *Eczema rubrum.*—The essential point about this variety of eczema is the hypostatic œdema, with high degree of vascular congestion. It commonly occurs in persons whose legs are covered with a thin, imperfect cuticle which offers little or no resistance to the escape of the plasma. The condition is aggravated by increased venous pressure (Varix). As a general rule ointments should be avoided. Paint the part three times a day with calamine lotion containing 1 per cent of boric acid. When all active exudation has ceased, and the surface has become dry and scaly, stimulating agents, such as tar and subchloride of mercury, may be added to the calamine lotion in place of the boric acid. When the skin is excessively œdematous and rotten, and especially if there be suppuration, it is well to anticipate the calamine lotion treatment by the use, for a few days, of Lassar's paste with 1 per cent of salicylic or boric acid.

Type 2. *Indurated chronic eczema behind the knees.*—Eczema has the same tendency to become chronic and indurated in this situation as in the bends of the elbows. The treatment is the same as for the elbows. (See Arms, p. 40).

Feet.—Exactly the same types are met with here as in the hands. There is, however, a greater tendency to intertrigo between the toes, on account of the accumulation of secretion, and the pressure exerted by the coverings of the feet. Horny eczema is apt to be more chronic than on the hands. After the plaster treatment an oleate of mercury ointment with beta-naphthol (10-30 grains) should be rubbed in vigorously two or three times a day. In very chronic cases, or in persons who have a past history of syphilis, a papillomatous hypertrophic

condition of the heel may develop, closely resembling tuberculated and syphilitic hypertrophy, but without the same tendency to spread beyond the regions of pressure. The cure is very tedious, but must be conducted on the same lines as those indicated above.

NAILS.—The nails, when involved in the eczematous process, become, like the palm cuticle, thickened and brittle, and lose their smooth, glistening aspect. The longitudinal ridges become coarse and pronounced, and the surface appears pitted "like the rind of an orange." In chronic cases the nail becomes greatly thickened. According to Mr. Jonathan Hutchinson eczema of the nail is to be distinguished from psoriasis by the fact that in the latter the process begins at the free margin, and not at the lunule, as in eczema. Further, in psoriasis the surface of the nail is unaffected, while the disease tends to the partial loosening of the nail. Arsenic, which in Mr. Hutchinson's experience cures psoriasis of the nails, has no effect on eczema in this situation, nor even on those mixed cases in which psoriasis and eczema coexist. Eczema of the nail is extremely rebellious to treatment. The best results in the writer's experience have followed the frequent local application of a strong solution of salicylic acid (ʒj.) in acetic ether (℥j.), the nail being scraped previous to each application. This treatment is not suitable if the surrounding soft parts of the finger are inflamed. The skin itself must be cured before the nail treatment can be undertaken.

The parasitic fungi, achorion and some varieties of the trichophyta are capable of invading the horny substance of the nail, which in such cases becomes brittle and tends to split into laminæ, in consequence of which it becomes rough and lustreless in appearance. Whenever the substance of the nail has a crumbled aspect, fragments should be softened in liquor potassæ and microscopically examined before pronouncing a diagnosis. If its parasitic nature were overlooked difficulties might arise on account of its contagiousness.

Eczematoid.—Resembling eczema, *e.g.* eczematoid dermatitis (*vide* DERMATITIS TRAUMATICA ET VENENATA, *Acute Eczematoid Dermatitis*).

Eddyism.—The system taught by Mrs. Eddy of Lynn, U.S.A., by which sickness is said to be cured by a mental process; Christian Science or Mind Cure. Her work, *Science and Health, with a Key to the Scriptures*, is said to have gone through 250 editions (1905). In 1866 Mrs. Eddy began to teach, in 1879 she organised the *Church of Christ Scientist*, and in 1876 her followers formed the *Christian Scientists' Association*. Eddyism comes into conflict (1) with medicine, for, according to its tenets, drugs, etc., are useless; and (2) with the law, where, for instance, a person has died while under the charge of a Christian Scientist who might presumably have been saved had ordinary remedial measures been applied.

Edebohls' Operation. *See* DECAPSULATION, RENAL.

Edocephalus.—That variety of the cyclopic monstrosity in which there is a nasal proboscis above the single eye, the ears are approximated or united under the head, the maxillæ are atrophic, and there is no mouth (*vide* Ballantyne's *Antenatal Pathology*, vol. ii. p. 425); it is derived from Gr. αἰδοῖον, sexual organs, and κεφαλή, head, the reference being to the penis-like proboscis above the single eye. *See also* CYCLOPIA; CYCLOTIA; TERATOLOGY; etc.

Education. *See* BLIND, TRAINING AND EDUCATION; MIND, EDUCATION OF.

Efferent.—Vessels, nerves, ducts, or strands of tissue which lead away from an organ or structure of the body; it is opposed to *afferent*. *See* PARALYSIS.

Effleurage.—A variety of massage movements, slow and gentle, given with the palm of the hand, from the periphery of the part toward the centre.

Efflorescence.—A rash or redness of the skin, also a group of cutaneous lesions (vesicular, papular, pustular, etc.) constituting one of the exanthemata. *See* MEASLES; SMALL-POX; etc.

Effluent.—A term applied, in connection with sewage, to the fluid part after the sewage has been subjected to purification; the purity of an effluent may be quickly tested by shaking it up in a bottle about half full for one minute, when, if the effluent be good, the froth should not remain for more than three seconds. *See* SEWAGE AND DRAINAGE.

Effusion.—As the derivation (*e*, out, *fundo*, I pour) indicates an effusion is something "poured out" into the subcutaneous tissue, into an internal organ or cavity, or on a free cutaneous or mucous surface; it may be blood, serum, lymph, chyle, or pus; it may occur as a transudate (in dropsy) or as an exudate (in inflammation).

Egersis.—Wakefulness of a marked kind (Gr. ἐγείρω, I awaken).

Egg. *See* DIET (*Eggs*); INVALID FEEDING (*Eggs*); PHYSIOLOGY, FOOD AND DIGESTION (*Animal Foods, Eggs*).—An egg will keep longer if it be coated with butter or varnish to exclude the air, or if it be placed in lime water with tartrate of potash; the freshness of an egg may

be tested by placing it in a 10 per cent saline solution when it should sink, but if it float more or less near the surface it is stale.

Egg Flip. *See* ALCOHOL.—Egg flip is an official preparation of brandy, and is known as *Mistura Spiritus Vini Gallici*; it is made by beating up the yolks of two eggs with half an ounce of sugar, four fluid ounces of brandy and the same quantity of cinnamon water are then added; the dose is from 1 to 2 fl. oz.

Egoism.—A state of mind in which the personal factor is predominant, showing itself in an excessive love of self or in an extraordinarily high opinion of self; opposed to altruism.

Egregorsis.—Morbid wakefulness (Gr. ἐγρήγορα, to watch).

Egypt. *See* THERAPEUTICS, HEALTH RESORTS (*Egypt*).

Egyptian Anæmia.—This disease is believed to be largely due to *bilharzia* infection, and less often to the *ankylostoma duodenale*; it is also known as Egyptian chlorosis.

Ehrlich's Reaction. *See* DIAZO-REACTION; LUNG, TUBERCULOSIS (*Complications, Uro-genital, Prognosis*); MALARIA (*Benign Tertian Fever, Urine*); TYPHOID FEVER; TYPHUS FEVER (*Period of Advance*).

Ehrlich's Theory. — Ehrlich's *Side-chain* theory, which gives an explanation of the general action of toxins. *See* IMMUNITY (*Cause of Antagonism of Antitoxin to Toxin*); PHYSIOLOGY, INTERNAL SECRETIONS (*Toxic Action and Immunity*).

Eighth Nerve. *See* BRAIN, PHYSIOLOGY OF (*Cranial Nerves, Eighth*); PHYSIOLOGY, NERVOUS SYSTEM (*Medulla and Cranial Nerves*).

Eiloid.—Like a coil (Gr. εἴλω, I coil up); *eiloides* is "a hypertrophic disease of the skin in which it becomes rolled on itself or folded" (*Syd. Soc. Lex.*).

Eilsen. *See* BALNEOLOGY (*Germany*).

Einhorn's Gastro - Diaphanoscope. *See* ENTEROPTOSIS (*Diagnosis*).

Ejaculation.—The ejection or emission of fluids (especially of the semen) from the body. *See* PHYSIOLOGY, REPRODUCTION (*Semen*); SCROTUM AND TESTICLE, DISEASES OF (*Sterility*).

Elæometer.—A hydrometer for determining the specific gravity of oils.

Elæoptens. — Liquid hydrocarbons, which, when mixed with oxidised hydrocarbons (usually solid bodies or stearoptens), form volatile or essential oils.

Elaidic Acid.—Isomeric with oleic acid; more stable than oleic acid.

Elapidæ.—Land snakes of the Colubrine class; *Elaps* is the "Coral snake" of the West Indies and Brazil. *See* SNAKE-BITES AND POISONOUS FISHES (*Classification*).

Elastic Tissue. *See* EXPECTORATION (*Microscopical Examination of Sputum, Lung Tissue, Yellow Elastic*); PHYSIOLOGY, TISSUES (*Connective, Fibrous, Elastic*).

Elastic Webbing. *See* ULCERS AND ULCERATION (*Ambulatory Treatment, Bandage*).

Elastin.—A substance nearly allied to the proteids; yellow elastic tissue consists of it; *elastose* is the product of the peptic digestion of elastin; it has a restricted value as a food stuff. *See* PHYSIOLOGY, TISSUES (*Connective, Fibrous*); PHYSIOLOGY, FOOD AND DIGESTION (*Food-stuffs yielding Energy, Proteids*); SKIN, ANATOMY AND PHYSIOLOGY (*Corium, Elastic Fibres*).

Elaterium. *See also* CATHARTICS; CONSTIPATION; HYDRAGOGUES; PHARMACOLOGY; PRESCRIBING.—The sediment collected from the juice of *Ecballium Elaterium*, the Squirting Cucumber. *Dose*—$\frac{1}{10}$-$\frac{1}{2}$ gr. From it is obtained the crystalline active principle, *Elaterinum*. *Dose*—$\frac{1}{40}$-$\frac{1}{10}$ gr. *Preparation* — Pulvis Elaterini Compositus. *Dose*—1-4 gr. Elaterium is the most powerful hydragogue cathartic in the Pharmacopœia. Its action depends entirely on the contained Elaterin, and is similar to that of Colocynth but more violent. Elaterium should contain from 20-25 per cent of the active principle, but specimens vary greatly in strength, and Elaterin itself is therefore more satisfactory. In chronic renal disease with dropsy, in ascites, and even in heart disease, Elaterium, by producing large watery evacuations, often proves most useful. It may, however, cause considerable collapse, and it should only be employed when other more mildly acting remedies have been proved inefficient. Patients differ greatly in their tolerance of the drug, and opinions vary considerably as to the indications for its administration.

Elbow - Joint, Region of, Injuries and Diseases of.

See also BRACHIAL PLEXUS, SURGICAL AFFECTIONS (*Arthritis of Elbow as Sequel*); BURSÆ, INJURIES AND DISEASES OF (*Situations of Bursæ, near Elbow-Joint*); OSTEO-ARTHROPATHIES (*Arthropathies, Charcot's Joints*).

Anatomical.—In the following description the term epicondyle will be taken to refer to that prominence of bone, on either side, which is without the capsule, and which may or may not be entirely epiphyseal.

The condyles proper, then, are within the capsule, intra-articular; the outer includes the capitellum, the inner the trochlea.

In the examination of all injuries about the elbow-joint a clear appreciation of the normal relationship of the parts is essential. The prominent tendon of the biceps is plainly visible in front of the joint. Its outer edge is separated from the extensor mass of muscles by a distinct furrow which indicates the line of the deeply placed musculo-spiral nerve. The furrow or hollow on its inner side, between the inner edge and the flexor group of muscles, is less visible and marks the situation of the median nerve and the brachial artery.

The fold of the elbow is a little above the line of the articulation (Treves). Its extremities correspond to the summit of the epicondyles.

The prominent and pointed internal epicondyle, the olecranon process, and external epicondyle are the important bony landmarks.

The summit of the olecranon is, in full extension, slightly above a transverse epicondylar line, but forms with this line in flexion a triangle, the apical angle of which varies with the degree of flexion.

The interval is greater between the olecranon and external epicondyle, and less important than that between the olecranon and internal epicondyle which lodges the ulnar nerve.

The natural deviation of the forearm outwards on account of the obliquity of the trochlear articular surface of the humerus should be borne in mind.

Immediately below the external epicondyle and just behind the muscular mass formed by the radial extensors and supinator longus, the head of the radius can be distinctly felt, indicating its point of contact with the capitellum.

On deep pressure in front of the joint the coronoid process can be felt (Chiene).

It is needless to add the advantage gained by a systematic and comparative examination of the injured and sound joint, and in various positions. Limitation of movement, of flexion, of extension, of pronation, of supination, and the existence of increased or diminished lateral deviation should be noted. By placing the middle finger and thumb of both hands on the epicondyles of the injured and sound joint respectively the forefingers can be placed on the olecranon, and its varying relation ascertained.

Care should be taken not to confound transmitted crepitation for that supposed to be under the examining finger.

Among the difficulties of diagnosis are the early rapid swelling of the parts, and the occasional presence of a more or less complete dislocation.

Fractures.—Fractures of the lower end of the humerus may be conveniently divided into:—

(*a*) Those which do not directly communicate with the joint.

(*b*) Those which extend into the joint and involve the articular surface.

(*a*) The only fracture that need be mentioned belonging to the first division is a fracture through both the epicondyles, in children practically including in the lower fragment the entire epiphysis.

This fracture is usually the result of an indirect blow either on the point of the elbow or on the hand.

It is most frequently met with in children (twelve out of eighteen—Hamilton).

The line of fracture in the adult is generally oblique; the direction of the obliquity depends upon the cause. Usually the lower fragment is displaced backwards, carrying with it the forearm.

The pointed ragged extremity of the diaphysis buries itself in the brachialis anticus.

The converse may be the case, the lower fragment with the forearm carried forwards and the diaphysis backwards.

In children the line of fracture is more transverse than oblique, involving a detachment, complete or in part, of the lower epiphyses.

Diagnosis.—All the signs of fracture are

present. Deformity is manifest, more so in the adult. Holding gently the epicondyles, they will be found to move with every movement of the forearm, and to permit of an amount of lateral movement which plainly establishes unnatural mobility.

With the fingers behind and the thumbs in front, the lower fragment can be guided into position. This movement may elicit crepitation.

Displacement immediately recurs, especially in the oblique variety, as the oblique surface forms no basis of support. The humerus will be found shortened on measurement.

If the injury be a compound one, the upper fragment, denuded of its periosteal lining, is usually shot through the skin. Whether in such an injury the joint is opened is a question much debated and all-important in regard to treatment and ultimate result.

That the joint is not opened in every case is certain, and even if opened may be effectually shut off from septic infection should such occur.

Occasionally the nerves, the musculo-spiral, median, ulnar, are damaged and sometimes torn through, necessitating operative interference. The brachial artery has been so injured as to give rise to gangrene.

Treatment.—One cannot emphasise too much the advantages of full anæsthesia in all injuries of the elbow-joint. It places the diagnosis on a more certain foundation, and renders accurate the apposition and retention of the fragments.

In this particular fracture, having reduced the displacement, some means must be taken to ensure fixation of the fragments.

Some use no splints at all, others fully flex the elbow and fix with bandages of sticking-plaster, making the triceps tendon act the part of a splint. A splint of some kind is probably advisable, but whether applied in front, behind, or laterally, in a position of flexion or extension, is a matter of opinion.

In the child the position of extension is irksome, and in the adult, if with traction, which is sometimes necessary, necessitates absolute confinement to bed.

A padded hoop-iron splint bent to the angle which seems most effective, and applied either to the posterior aspect of the limb alone or to the front as well, answers admirably in children.

In the adult, if extension with traction be found most serviceable, care should be taken that union does not occur with the forearm in a position of varus or valgum, a result more likely to happen in treatment by extension than in treatment by flexion.

A posterior moulded splint applied with the forearm in a position of full flexion seems in our opinion to yield the best results.

No splint should be kept on longer than three weeks, when passive motion should be commenced.

(*b*) Of the fractures of the lower end of the humerus which directly communicate with the joint, exclusive of epicondylar fractures, there are two chief varieties :—

1. Oblique fracture through the articular end separating the capitellar surface ; and

2. Oblique fracture through the articular end separating the trochlear surface.

1. By far the most frequent variety is that involving the capitellum.

The usual line of fracture is from above, downwards and inwards from a point immediately below the external epicondyle to the ridge separating the capitellar from the trochlear surface, though a varying amount of the capitellum may be carried away.

It is mostly met with in young persons up to the age of puberty, and may, like all other injuries to the elbow, be the result of a direct blow on the elbow itself. It is usually, however, the result of a fall on the pronated hand, so that the force is transmitted through the radius to the capitellum, which is thereby driven upwards, carrying with it in some cases the external epicondyle.

Diagnosis.—On the arm being straightened the fragment is pulled downwards by the external lateral ligament, and rotated outwards, so that in some cases it can be felt under the skin.

The elicitation of crepitation may be difficult owing to separation of the fractured surfaces, and specially so if the fracture be accompanied by a dislocation backwards of the radial head.

Any attempt at movement causes intense pain.

The results of treatment hitherto of this fracture have not been satisfactory. If union occur with the fragment displaced and rotated, excessive callus is thrown out, causing subsequent limitation of movement and an awkward projection in front of the joint; and, on the other hand, if the fragments do not unite, such an amount of cubitus valgum results as to render the arm remarkably useless.

Though in children continued passive motion may improve matters, perfect movement is impossible, and a varying amount of valgum will remain.

Treatment.—Opinion varies as to the best line of treatment.

No one doubts the value of full anæsthesia in an attempt to fix in position the broken fragment, but whether this can be done effectually without cutting down and wiring or pinning the fragment is a question still *sub judice.* That this minor operation can be easily and safely done there is no question whatever.

Without operation the difficulty lies in replacing and keeping the fragment in position. A padded posterior metal splint where the angle can be changed every few days, with local pressure over the fragment by a pad, will reduce the chance of ankylosis or of vicious union.

Transverse intra-articular Fracture of the Capitellum.—The capitellum may itself be fractured without involving the external epicondyle, the fragment usually being completely separated and lying free in the joint.

Very much the same cause acts as in the oblique variety, viz. fall on the hand with force transmitted through the radius.

The signs are also similar; the head of the radius appears subluxated; the detached fragment may be clearly made out.

The movements of full extension and supination are limited and painful.

The removal of the separate fragment through an incision on the outer side of the joint between the biceps tendon and the supinator longus forms the only satisfactory treatment.

2. Oblique fracture through the articular end separating the trochlear surface is less common than separation of the capitellar surface, and less likely to be troublesome.

The displacement is less, and the position of the fragment more easily maintained.

As a further complication of this oblique fracture is longitudinal splitting of the shaft of the humerus—the T or Y fracture.

These are more severe injuries, and not only involve the joint, but are frequently compound.

The lower end of the humerus is widened transversely, and either fragment can be moved apart from the other and from the shaft.

The same remarks apply as to treatment. Conservative measures should be adopted if the vessel escape; a subsequent excision is better than a primary amputation.

Fracture or Separation of the Epicondyles:—

I. Fracture of the internal epicondyle.
II. Fracture of the external epicondyle.

Of the fractures of the epicondyles separation of the internal epicondyle is by far the more common.

In the adult it cannot be said that the line of fracture follows the line of the epiphysis; whether the shaft is involved or not largely depends upon the cause.

Up to the twenty-first year it is otherwise, and may be regarded as a separation of the epicondylar epiphysis.

This injury is usually the result of a direct blow on the epicondyle itself, but may be the result of traction, either through the sudden and violent contraction of the muscles, flexors, and pronator (Granger), or through the internal lateral ligament, the result of forcible abduction of the forearm (Helferich).

Whatever the cause, the separated fragment is almost invariably drawn downwards, and perhaps also slightly forwards and outwards.

If the ulnar nerve has escaped primary injury this situation of the fragment lessens the chance of a future disturbance of its function. Cases have been recorded, however, where the displaced fragment has had to be subsequently removed on account of nerve pressure.

The unnatural mobility of the displaced fragment can be readily demonstrated; there is usually some extravasation of blood into the joint. Union is always fibrous and in the position of displacement.

No means short of pegging or pinning the fragment have been successful in keeping it in position, and though this is recommended as the only certain line of treatment, there is rarely much permanent ankylosis or limitation of movement if the joint is not kept too long fixed. Passive motion should be practised early between the second and third week.

Fracture of the external epicondyle alone is very rare.

The same remarks apply to this fracture as to the other.

Fractures of the Radius and Ulna in the Region of the Elbow-Joint

Fracture of the Olecranon Process.—This fracture may result from direct violence, forcible contraction of the triceps, or from hyperextension. The first of these causes is by far the most frequent.

The process may be broken at its base or at its apex, though it is most frequently fractured about its centre, where it is narrowest; the usual direction of the fracture is straight across.

Up to the age of seventeen the line of fracture may correspond to the line of the epiphysis, practically the apex of the process.

The amount of separation of the fragments depends upon the extent of laceration of the periosteal covering and the lateral triceps aponeurosis; the more these are torn the greater the separation.

The diagnosis of this injury is not difficult if seen sufficiently early. The subsequent swelling is usually considerable, so that the gap between the fragments may only be felt with difficulty.

By approximating the fragments in many cases the gap can be made to disappear and crepitation obtained. Care must be taken not to confuse the crepitation which is sometimes present in an inflamed bursa, or to hurriedly conclude that no fracture exists from the apparent absence of crepitation.

The circumstances must be favourable to obtain bony union; there must be little or no separation of the fragments, which means little or no damage to the periosteum or fibrous aponeurosis.

Union is almost invariably fibrous, sometimes one band, sometimes two connecting the fragments.

The parts should be fixed by an anterior splint, with the elbow fully extended, and the fragments approximated by strapping or by figure-of-eight bandage.

There is little danger of ankylosis if massage be practised from the end of the first week, and passive movement carefully performed whenever union is apparent.

Bony union may be secured by wiring, but since firm fibrous union gives a satisfactory result, it is questionable whether an operation is justifiable, unless under the most favourable circumstances.

Fracture of the Coronoid Process.—This is uncommon as a separate lesion. It is most often met with as an occasional accompaniment of dislocation backwards of both bones of the forearm.

Only if the fracture is at the base of the process can the brachialis anticus exert any action.

If the tip be alone separated one would presume the union to be fibrous; osseous union is possible at the base.

Treatment consists in fixing the limb in the flexed position.

Fracture of the Head of the Radius.—Fracture of the head of the radius alone is rare.

The force necessary to fracture the head of the radius spends itself on the neighbouring projections, the capitellum, the olecranon and coronoid processes, so that fracture of one or other may accompany fracture of the radial head.

The cause may be direct or indirect, the fracture complete or incomplete. The diagnosis in the complete form rests on mobility of the detached fragment, with crepitus. In the incomplete form the difficulty of diagnosis lies in the fact that only a part of the head may be involved, and the fragment immobile owing to the security gained by the unruptured annular ligament.

As would be expected from the entirely intra-articular situation of the fracture, there is immediate extravasation of blood and subsequent effusion into the joint, with pain and crepitus on rotation, when the smooth radial head will be replaced by an angular projection.

To secure union of the fragment fixation in the flexed position offers the best result from expectant treatment, but ankylosis is apt to occur, and seems to justify an attempt to remove or pin in position the loose fragment, premising that absolute asepsis can be attained.

The author has in his museum an exact half of the head of the radius broken off by a fall on the elbow and removed by operation, with subsequent perfect movement.

Fracture of the Neck of the Radius.—Fracture of the neck of the radius as a separate injury is also rare. The diagnosis depends upon the elicitation of crepitus and the fact that the head of the bone does not move on rotation of the forearm.

The biceps, acting on the tubercle, draws forward the lower fragment; the orbicular ligament may keep the fragments in position.

A posterior or internal rectangular splint with a pad over the upper end of the radius gives the best result.

Dislocations at the Elbow-Joint

A dislocation of both bones is more commonly met with than a dislocation of either the radius or ulna alone.

Of the dislocations of both bones, that backwards is the most frequent.

Dislocation Backwards of both Bones.—Any cause likely to hyper-extend the forearm when the arm is more or less fixed would tend to produce this form of dislocation, *e.g.* a fall on the outstretched hand.

A violent twist of the forearm and a blow either on the upper and front aspect of the forearm, or on the lower and hind aspect of the arm, have much the same effect.

Both bones are carried backwards, with rupture of the anterior and lateral ligaments, and drawn upwards by the triceps.

The orbicular ligament usually escapes injury and assists in keeping the bones of the forearm in their normal relationship, so that though the coronoid process is opposite the olecranon fossa, it is prevented from entering it by the radius, and thus the extent of upward dislocation varies.

The tendons of the biceps and brachialis anticus suffer.

The median and ulnar nerves may also be injured.

Some of the surrounding bony projections may be fractured, notably the coronoid process, and occasionally the internal epicondyle and olecranon.

If seen before much swelling occur, the diagnosis is comparatively easy. The forearm will be found fixed in a position of semiflexion, and shortened. The olecranon will be found unduly prominent, increasingly so if flexion be attempted, and quite above the epicondyles; the head of the radius visible and tangible; the humerus is normal in length, altered in axis, and projects, though deeply placed in front.

Reduction should be effected by hyper-extending the supinated forearm, so as to lift the coronoid process free of the olecranon fossa, followed by traction and gradual flexion, at the same time guiding the olecranon, and steadying the lower humeral end with the disengaged hand.

It is better, at any rate safer, to fix the elbow with a splint for a fortnight or so than simply to rely on the sling alone.

In old unreduced dislocations it is often impossible to effect reduction, even with the aid of pulleys. If movement be much impaired, and the resulting false joint ineffective, a free exposure of the opposing bone surfaces, by division of the various ligaments and bands of fibrous tissue, will, with very few exceptions,

allow of reposition of the bones, and give a better result than resection.

That this may be done satisfactorily, nine or even twelve months after the injury, has been proved by the author in several recent cases.

Dislocation forwards of both Bones.—A subluxation forwards of both bones is very rare. The comparatively large and curved olecranon process prevents luxation forwards, and it is only when the olecranon is taken at a disadvantage, as when the forearm is fully flexed, that luxation is possible without fracture. The injury to the soft parts is considerable.

In effecting reduction, the arm should be fixed while traction sufficient to bring the olecranon over the trochlea is made; the forearm is then flexed.

Lateral Dislocation of both Bones. — Of the lateral dislocations, that outwards is the more common, possibly on account of the slanting trochlear surface of the humerus. Both are uncommon, and usually incomplete.

In the outward variety the internal lateral ligament is torn, or the internal epicondyle separated.

The trochlear humeral surface can be readily felt and defined; the capitellum is embraced by the sigmoid; and the radial head lies quite beyond the external condyle, where it also can be felt and seen projecting just under the skin.

In the inward variety the reverse condition is met with. The external lateral ligament is ruptured, or the external epicondyle separated. The head of the radius rests on the trochlear humeral surface, while the sigmoid, with its olecranon and coronoid processes, can be felt surrounding the internal epicondyle.

There may be some backward or forward displacement of one or other bone in addition.

Reduction in either form is best done under an anæsthetic, and might be expressed thus — hyper-extension, lateral pressure, traction, flexion.

Dislocation of the Radius or Ulna alone.— One or other bone may alone be dislocated, or one bone may be dislocated forwards and the other backwards.

Of the two the radius, from its position and shape, is the more usually involved, and may be dislocated forwards, backwards, or outwards. Dislocation forwards is the more common variety, and comes next in frequency to dislocation of both bones backwards.

Dislocation forwards of the Radius. — The commonest cause of this dislocation is a fall on the extended and pronated hand; occasionally it is the result of a blow on the upper part of the radius itself from behind.

The surrounding ligaments are torn; the orbicular may escape in the child, but suffers like the others in the adult.

The displaced head of the radius can be felt lying to the inner side of the supinator longus and the extensor muscles, and to move when the forearm is supinated or pronated.

Complete flexion of the forearm is impossible.

In reduction fix the humerus and employ traction with the forearm flexed and supinated, extend, and with the thumb of the hand grasping the humerus guide the head into its place.

If attempts at reduction are futile, perhaps from an intercepting piece of torn capsule, the joint should be explored by a lateral incision and the cause removed.

Note. — This dislocation is a not infrequent accompaniment of fracture of the ulna in its upper third, and while the fracture is easily recognised, the dislocation may be overlooked (Helferich).

Of the other dislocations of the radius little need be said; both the backward and the outward forms are infrequent, and not difficult to diagnose.

In young children, as pointed out by Jonathan Hutchinson, jun., and others, a sudden and violent pull to the forearm, especially if supinated, is apt to cause the radial head to slip the orbicular ligament. There is little or no deformity at the elbow as a result, but the arm hangs helplessly by the side, and is very painful to manipulate.

With flexion and full pronation the head will return to its place.

Dislocation of the Ulna alone (uncommon).—If the radius, from its position and shape, is more frequently luxated forwards, the ulna, for similar reasons, is more frequently luxated backwards.

This dislocation closely resembles on superficial examination a dislocation backwards of both bones, but close examination reveals the normal situation of the radial head.

The ulna is easily replaced.

Sprains

A sprain of the elbow-joint differs in no respect from a sprain of any other joint, except it be the fact that if the wrench be at all severe it is usually not a simple sprain but accompanied by separation of one or other of the bony prominences.

The ligaments of the elbow-joint, with the exception of the anterior and posterior, are very strong, and though some strands of fibres are torn, it is exceptional to have their complete rupture.

The tendinous and aponeurotic attachment of the various muscles around the joint are stretched, and in places torn, causing rupture of the smaller blood-vessels and extravasation of blood.

Subsequent ecchymosis indicates the site, and, to a certain extent, the severity of the sprain.

The synovial membrane shares in the damage done, with the result that free hæmorrhage may take place into the joint. The nerve-trunks may be implicated from pressure.

The diagnosis of a simple sprain is arrived at by a process of exclusion. The rapid swelling in and around the joint, due primarily to hæmorrhage and afterwards to effusion, makes it difficult at once to express a decided opinion. The relationship of the bony prominences to one another, and with the sound joint, the presence or absence of limitation of movement, of extension and flexion, of pronation and supination, should be ascertained.

It is best, if in doubt, and especially in children, to give a guarded opinion, as separation of the epiphysis may readily be mistaken for a simple sprain, and the mistake only found out when growth is arrested.

Treatment in the early stage resolves itself into giving the joint complete rest, and arresting the internal hæmorrhage.

For both a splint is essential, and for the latter pressure if it can be tolerated. The joint should be surrounded with cotton-wool and an elastic webbing bandage applied.

Cold in the form of ice-bags and Leiter's tubes has been much vaunted, but does not seem to be so comforting or efficaceous as supposed.

If the effusion has apparently reached the limit, and be accompanied with considerable tension and pain, fomentations, medicated or otherwise, give great relief, as does also aspiration if it can be judiciously carried out. When the effusion is beginning to subside, and organisation with absorption commencing, it is then that much care should be exercised to prevent adhesion and fibrous ankylosis resulting.

Passive motion with massage and douching should at this stage be daily practised, being guided by the presence of local reaction to its continuance.

Should firm adhesion have already occurred, the joint can be moved under an anæsthetic.

Rupture of muscular fibres, as in the case of "Tennis Arm," where the pronator teres is partially torn, and even of the biceps tendon from forcible flexion, have been noted, both being indicated by the usual signs of such injuries.

Wounds

Wounds in the neighbourhood of the elbow-joint may, as in the case of fractures, be divided into :—

(*a*) Those which do not communicate with the joint; and

(*b*) Those which directly or indirectly communicate with the joint cavity.

Wounds which do not communicate with the joint resemble in their classification and treatment wounds in general.

It is to the injury done to important structures, and the possibility of direct septic infection, that attention should be paid.

One or other of the large nerves may be divided, or the brachial vessel damaged. The chance of sepsis, of course, is more likely to arise if the wound be a punctured one.

Where the nerve has been divided, and when the circumstances are favourable, an attempt should at once be made to suture the divided ends. Failure to recognise or to suture at the time a divided nerve would render necessary a subsequent operation.

In a cleanly cut wound where the tissues have not been lacerated or contused, and where, as far as can be ascertained, no infective material has been introduced, the divided structures should all be brought together.

Attention should also be paid and care taken to prevent or limit contraction of the scar, especially in the case of burns or scalds. Cicatricial tissue over the olecranon is very liable to break down, and a healthy flap or graft should be substituted.

It is, however, when the wound communicates with the joint that special precautions have to be taken, and a more guarded prognosis given. The fact that a given wound does communicate with the joint is not always easy to demonstrate. The wound of entrance may be a punctured one and at some distance from the joint, the immediate swelling of which would certainly be suggestive of its involvement, as would also the escape of synovial fluid, though it is to be remembered that this may be bursal.

The particular danger lies in septic infection of the joint, resulting in the possible necessity of amputation, though this is very seldom required in the case of the elbow, or at best of ankylosis or of resection.

If the wound be a simple one and heal kindly, the function of the joint is not disturbed. Aspiration should at once be made if there be evidence of local heat or tenderness; the presence of sero-pus would point to a free opening of the joint and thorough irrigation.

The joint is best opened at the outer side of the olecranon, though more than one opening may be necessary.

Where the damage done, however, is extensive, involving comminution and splintering of the bones, the question of primary resection or of removal of the damaged fragments should be considered.

Septic wounds involving the olecranon bursa are always serious, and call for free incision into and cleaning of that cavity lest a secondary suppurative periostitis leads to involvement of the elbow-joint.

Gunshot Wounds.—Gunshot wounds involving the elbow-joint are comparatively rare in civil practice. Their prognosis and treatment depend upon the extent of bone implicated and the amount of damage done to the surrounding soft parts. As in other wounds of this joint, unless the parts are irretrievably damaged, conservative measures should be tried.

Should the bones have escaped injury, a some-

what exceptional state of affairs, measures should at once be taken to keep the parts aseptic. The joint should be fixed at right angles with the forearm, midway between supination and pronation, and an attempt made to secure subsequent movement.

If the bones have been injured, and possibly comminuted, any loose and projecting fragment can be removed and the joint irrigated and fixed.

Where the articular surfaces are implicated, ankylosis is an unavoidable result, and care should be taken to ensure that the best position is got, which will probably be found to be a little less than a right angle.

It is perhaps better at this stage only to remove what is hopelessly damaged than to perform a classical excision.

Should conservative measures fail and suppuration ensue, with death of the injured fragments, a secondary operation can be performed.

Primary amputation is only required where destruction of the bones is very extensive, and where the main vessel is torn across.

Contractions.—Destruction of the skin by injury, as in burning or disease, induces contracture of the joint from cicatrisation.

Means must be used to counteract this tendency by early skin grafting, preferably with large grafts or pedunculated skin flaps from the arm above or below, or, as recommended by some, from the chest. In the minor degrees of contracture continuous extension may be sufficient (Schede), but in the more severe forms it will generally be found necessary to divide the cicatrix transversely after forcible extension, and graft the exposed surface.

Myogenic contraction may cause stiffness so great as to necessitate treatment. It may follow injury or disease, especially syphilitic disease of the biceps, or be the result of spasm (of biceps, Stromeyer) due to altered nerve function.

Ankylosis.—Ankylosis of the elbow may result from tuberculous or suppurative disease of the joint, and necessarily interferes much with the usefulness of the limb. When the forearm is fixed at somewhat less than a right angle the limb may be fairly useful, at least more so than when straight, though in both cases the limb may be useful for particular purposes. When straight the limb can only be used in some handicrafts.

Following tuberculous disease, and especially in young children, it is well to wait until the arm is nearly fully developed before excising, as considerable distortion of the limb may result from corresponding diminution of its usefulness.

Partial ankylosis may take place. In union at the radio-ulnar articulation, with limitation of pronation and supination, either from fracture or from disease, excision of the head of the radius, or merely dividing the bond of union, may be sufficient, provided attention be paid to subsequent movement.

Prolonged fixation after injury may lead to partial ankylosis from shrinkage of the capsule or formation of fibrous adhesions.

The treatment consists in immediate and forcible stretching of the resistant structures under an anæsthetic or, according to the degree of resistance, by gradual extension from weights or mechanical aids such as may be obtained by an adjustable hinge-joint splint.

A plaster of Paris splint applied to the arm with metal hinges opposite the joint, and the plaster cut away from before and behind the joint, is useful, as by means of pieces of cork inserted between the edges of the plaster, force may be gradually brought to bear so as to correct the contraction : elastic traction may be used instead of cork.

Other causes of limitation of movement in the elbow-joint are deformity of bone in osteo-arthritis, and thickening of the bone in periostitis, leading to filling up of the fossæ or to expansion of the bony processes.

Cubitus Varus and Valgus, the former being abnormal flexion and pronation of the forearm, and the latter excessive supination and radial flexion of the forearm, may be due to fractures or rachitis, or be in some cases congenital. Osteotomy will rectify the deformity in some cases of the acquired form; as a rule no treatment is required in the congenital variety.

Inflammatory Diseases.—In the diagnosis of all affections of the elbow-joint it will be found that inspection and palpation of the posterior part of the joint by the sides of the olecranon are especially important, since it is there that any abnormal fulness of the joint will be first noticed.

The head of the radius can be examined by palpation aided by movements of pronation and supination.

Flexion and extension are early restricted in all diseases of the elbow-joint, and, as a rule, the limb becomes fixed in a position of flexion and semi-pronation, specially if there be effusion, since in that position the joint has the greatest capacity.

Reflex contraction of the biceps aids the flexion particularly in acute affections.

Simple Synovitis may result from injury or exposure, and is characterised by a sense of stiffness and pain, especially on movement.

The arm is held flexed and semi-pronated, while soon there appears swelling on either side of the olecranon, due, as may be determined by palpation, to the accumulation of fluid in the joint.

The treatment consists in securing absolute rest by an angular well-padded splint applied to the inner aspect of the limb, relaxing the tension of the part by the use of antiseptic fomentations, and a saline purge preceded, if necessary, by a dose of calomel.

In asthenic cases leeches have been used with advantage.

Should the effusion become considerable, aspiration from one of the prominent pouches by the side of the olecranon will give relief and may be repeated. After the acute stage passes off, massage, elastic compression, and passive movement may be continued till the stiffness has passed off.

Acute Septic or Suppurative Arthritis is characterised by the addition to the symptoms of simple synovitis of great œdema around the joint and for a considerable distance from it, of the redness of the skin, rigors, and of high fever, with the usual profound constitutional disturbance attending marked septic intoxication.

In such cases the treatment must be active and immediate.

Free incisions on each side of the olecranon will enable the joint to be irrigated with an antiseptic solution, while the limb is immobilised on an angular splint at an angle somewhat less than 90°.

Since ankylosis in such a case is to be feared, it is well to have the arm in that position in which it will be most useful when fixed. Drainage of the joint will require to be continued till the acute process has subsided, and, should this take place in a few days before disorganisation of the joint has occurred, it may be permissible to aim at securing movement by altering the position of the limb occasionally so as to stretch adhesions.

Early free movements of the joint are to be avoided as tending to keep up the inflammatory mischief.

Only when such can be practised without causing much pain is good likely to accrue, since only then has the inflammatory action subsided. Massage and douching, with passive movement continued over a long period, will in some cases give a movable joint, but in the great majority of cases the suppuration is attended with such a disorganisation of the joint that in healing ankylosis is a necessary result.

Scarlatinal and Rheumatic Arthritis differ in no respect from a similar condition in other joints; the local treatment is that of acute and subacute synovitis, with special attention, however, to the constitutional dyscrasia.

Gonorrhœal Arthritis of the elbow presents the usual features of an acute or subacute synovitis, and, as usual, is not amenable to treatment until the urethral trouble is rectified.

Subacute Synovitis is less commonly met with in the elbow than in some other joints, and presents the usual characteristics — the effusion, aching pain, and thickening of the synovial membrane and capsule.

The treatment consists of rest with the use of blisters, or, if much effusion exists, of aspiration, elastic bandaging, and massage.

In the more chronic forms it has lately been recommended to incise the joint and dissect away as much of the thickened synovial membrane as possible, after which the joint is to be washed out and the wound closed without drainage if possible (Treves).

Chronic Inflammatory Diseases. Tuberculous Disease.—This disease is by far the most common and important one of the elbow. Of all cases of tuberculous joints it may be roughly estimated that about 7 per cent are of the elbow-joint.

The disease may be met with at all ages, though it is usually met with in early life between the ages of three years and puberty.

The disease is, however, not uncommon in old people, in whom it is characterised by a tendency to progress unfavourably, and often rapidly so. The mischief may be primarily synovial, but in the elbow is less commonly so than in other joints. Statistics suggest that in about three-fourths of the cases the tuberculous focus will be found in one or other of the bones entering into the formation of the joint. The olecranon is a frequent seat of primary mischief, next in order is the humerus, while last and much less frequently affected is the head of the radius.

In early life, before the age of five, and in old age the disease is usually primarily synovial.

The bone affection may be recognised by the formation of an enlargement and some tenderness at one part of the bone, and later, as the tubercle approaches the surface, by the skin becoming œdematous, tuberculous, or it may be by a localised abscess.

Early evacuation of such a tuberculous focus may prevent the joint becoming involved.

When the general synovial membrane becomes affected an unmistakable clinical picture is presented which is most marked in the young, and is characterised by the following signs and symptoms.

In the early stage there may be little complaint of pain, attention being drawn by an increasing swelling and stiffness of the joint.

The swelling is most noticeable by the sides of the olecranon and varies within wide degrees. In children it is as a rule most marked, and gradually masks all the bony prominences in a diffuse globular swelling. This swelling, when occurring with great rapidity, is usually due to synovial effusion, ultimately it is due to the pulpy thickening of the synovial membrane. In acute cases the swelling is increased by œdema of the superficial structures, by the formation of tuberculous foci outside the capsule of the joint, and it may be by the formation of a chronic or subacute abscess in or in the vicinity of the joint.

In those cases where the disease is primarily osteal there may be corresponding enlargement of the bone with alteration in the configuration of the joint.

Swelling is often not at all a marked feature

in the old or even in young adults, and may indeed be so slight even in old-standing cases as to lead the surgeon astray as to the existence of tubercle in the affection; such cases usually present marked features of bone involvement.

Pain.—In the early stages there may be little or no pain even on gentle movement. Generally it may be elicited by palpation, and, in any case, is complained of after the use of the limb for some time, and is of course always a marked feature when early ulceration of cartilage takes place. As a symptom of tuberculous disease of the elbow, pain is of uncertain value.

Stiffness.—As the disease progresses gradual flexion to an angle of 140° or thereby takes place. Extension is early interfered with, though other movements may persist for a considerable time. Ultimately the joint gets more and more fixed, and if left alone osseous or fibrous ankylosis ensues, generally at an angle of 140°-145°, and with the hand in extreme pronation.

Wasting of Muscles. — This is especially marked in the upper arm, is very constantly met with, and accentuates the swelling in the elbow.

If the case be allowed to run its course, abscesses or intra-articular suppuration lead to the formation of sinuses usually most marked on the outer side of the joint.

The constitutional manifestations are those of localised tubercle. (*See* "Bones" and "Joints.")

The treatment of tuberculous disease of the elbow-joint differs not only according to the age of the patient, the type of the disease, and its stage, but according to the social position of the patient, and the possibility of subsequent good hygiene and dietary.

In healthy subjects, when the disease is seen early, and especially in the young, even although the disease may be severe, it may be arrested by appropriate local remedies and constitutional treatment, and the joint be left in a useful condition.

The local remedies applicable to the elbow are those used in joints elsewhere. In the stage of effusion a 10 per cent sterilised iodoform emulsion (glycerine or oil), repeated every fourteen days or so, will be found most useful. The injection is best made by the side of the olecranon, and the joint ought to be freely moved after the mixture is injected that it may be diffused over the whole joint surface. Twenty to sixty grains of iodoform may be injected at one time. The limb must be fixed in a suitable splint and gentle massage of the joint practised daily. Good results have also been obtained by the hyperæmic engorgement method of Bier.

Tuberculous abscesses must be treated as they form with the usual antiseptic precautions.

These measures, with constitutional treatment, may result in the recovery of the joint with its functions but little impaired.

In the majority of cases, however, operation will be called for, and the aim of this, as in operation for the other manifestations of tubercle, is to remove as much of the disease as possible, and yet to leave a functionally useful joint.

When the disease is primarily synovial, erasion of the joint rather than excision is to be preferred, especially in the young, even in those cases where deposits of tubercle exist in the bone, as these may be scooped out.

If asepsis has been secured, complete recovery may ensue with a useful joint.

Should the disease have been primarily osseous, or have involved the bones deeply by extension from the synovial membrane, then it is better to perform the classical excision.

Whether erasion or excision be performed, it is absolutely essential that as much of the disease as possible should be removed, the synovial membrane entirely dissected away, implicated bursal extensions cleared out, secondary nodules without the capsule searched for and evacuated, and foci in the bone cleared out by the gouge or spoon.

Syphilitic Arthritis is not uncommon, especially in the case of hereditary syphilis in children. The disease simulates closely tuberculous arthritis, and it is extremely probable that much of the good accounted to Scott's dressing (Ung. hydrarg.) in that affection is due to mistaken diagnosis.

In the early forms of the disease there is well-marked serous effusion into the joint, while later there appears localised thickening of the synovial membrane from the formation of gummata, or a general thickening from interstitial inflammation.

In doubtful cases the use of mercury and iodide of potassium will clear up the diagnosis.

Chronic Rheumatism, Gout, Osteo-Arthritis, Charcot's Disease, are all met with in the elbow. A detailed description of each will be found under their respective headings; the pathological and clinical features are similar to those of a like affection of other joints, treatment in the case of the elbow differs in no respect.

Hæmophilia. — Cases have been reported where in this disease hæmorrhage has suddenly taken place into the joint with swelling and pain. The swelling gradually subsided, leaving the joint stiff and to some extent disorganised.

Bursitis.—The following bursæ are found in the region of the elbow-joint:—(1) Over the olecranon; (2) beneath the tendon of the triceps muscle; (3) over the epicondyles; (4) upon the tuberosity of the radius beneath the insertion of the biceps tendon; (5) between the radius and ulna below the articulation.

Acute inflammation usually follows a septic wound of the bursa, that over the olecranon being most frequently affected.

Occasionally a hæmatoma of the bursa may

terminate in acute suppurative inflammation; in such cases the joint may be involved in the suppurative process, especially in old and debilitated patients, by the bone becoming affected either from a suppurative periostitis opening into the joint directly, or by inducing necrosis of the olecranon.

In other cases diffuse cellulitis of the arm may follow owing to the intimate communication of the bursa with the lymphatics.

Chronic bursitis may result from irritation due to occupation, and be either characterised by serous effusion or with considerable thickening of the bursal wall. More frequently the chronic form of the affection is due to syphilis, gout, or tubercle, in all of which the thickening of the bursal wall is, as a rule, a marked feature.

Diagnosis.—Bursal affections are characterised by the occurrence of well-defined swellings, usually fluctuant, in the site of one or other of the bursæ. The acute inflammatory forms soon, however, lose the circumscribed character from the surrounding inflammatory transudation or acute cellulitis, but the situation of the infected wound or the history may indicate the true origin of the mischief.

The treatment of the different forms of bursitis varies with the cause. In the suppurative form early and free incision, with frequent antiseptic packing of the wound, will best avoid the risk of extension to the surrounding parts. The syphilitic and tuberculous forms are best treated by as complete an excision of the sac as may be possible. In the deeper-seated bursæ the use of a Volkmann's spoon may enable the greater part of the disease to be removed with the least disturbance of the surrounding parts.

Hæmorrhage into the bursa may be treated by rest and elastic compression, or should there be undue delay in absorption, by excision and evacuation under careful antiseptic precautions. (*See* "Bursæ," vol. ii.)

Blood-vessels.—Aneurysm may result from venesection, and was formerly not uncommon. Due to wound of the brachial artery near the bend of the elbow, the result is either an ordinary traumatic or arteriovenous aneurysm, which gives rise to marked circulatory disturbance in the forearm and hand. The treatment consists in proximal and distal ligation of the artery with excision of the sac; should operative interference be inadvisable, by compression of the arm above the aneurysm or by forcible flexion of the elbow. (*See* "Aneurysm," vol. i.)

Inflammation of the veins and lymphatics in this region only occurs as the result of wounds or operations when antiseptic precautions have not been taken.

Neuralgia of the Elbow may result from an inflammatory affection of the parts, or be due to mischief farther up the limb; thus, pain about the elbow is occasionally complained of in diseases involving the armpit or shoulder. Pain may also result from local nerve injury. Dislocation of the ulnar nerve may take place from a blow or, apparently in some cases, without any definite cause, in which case it has been supposed that the displacement has been facilitated by a want of development of the internal condyle.

The nerve slips over the condyle in flexion, and may also be readily placed in front of the bone by manipulation. Considerable pain and reflex loss of power may result, necessitating fixation of the nerve in its place by stitching over it a flap of deep fascia, and in some cases deepening the groove in the posterior surface of the bone.

In the majority of the neuralgic affections of the elbow some definite cause of irritation may be found, but in some neurotic individuals of debilitated constitution shooting pains of a true neuralgic character may occur without a known cause. In such cases it is well to encourage free use of the joints, while a tonic course of general treatment and the local application of liniment of iodine, blisters, or even of Corrigan's button, will often give relief.

New Growths about the elbow present no special features of consequence. Osteal and periosteal tumours are most frequent in early life. Sarcomata, chondromata, and osteomata may occur in the bones of the elbow-joint, and require excision or amputation according to the nature and extent of the tumour.

Loose Bodies are met with less frequently in the elbow than in some of the other joints. When present they give rise to pain and limitation of movement, and may usually be readily recognised by palpation. They cause crepitus on movement. Treatment consists in their removal under antiseptic precautions.

Excision of the Elbow-Joint

This operation may be required for disease or injury. Of the former, tuberculous disease forms the greatest proportion; and of the latter, gunshot wounds and unreduced dislocation. It may be necessary also to excise the joint for ankylosis if union be in a faulty position.

1. Excision of the Joint.—The line of incision is better longitudinal than transverse, as a transverse incision needlessly damages the triceps aponeurosis and gives no better exposure of the joint. The posterior median incision is preferred by most; the lateral incisions of Ollier and Hueter are complicated and seem to have no special advantage.

Some lay considerable emphasis on the preservation of part of the attachment of the anconeus, separating it from the ulna no farther than a point just below the base of the olecranon. It is said to undergo development subsequently and to assist greatly in the extension of the forearm.

Injury to and division of the ulnar nerve is

regarded as the chief danger of this operation. The nerve should not be seen.

It lies between the olecranon and the internal condyle, and is retracted with the other structures. There is no danger if the operator keeps close to the bone in separating the tissues from the internal epicondyle and from the posterior ridge of the ulna.

The posterior interosseous nerve has been injured in clearing the head of the radius.

If excision be performed in the child the epiphyseal line should be preserved in the removal of the articular humeral end.

Excision through a posterior Median Incision. (Park and Maisonneuve.) — In regard to the position of the surgeon and his assistant the same rules hold good as in operations in general.

The forearm of the affected limb is best carried across the chest. Though some deprecate the use of the tourniquet, its application has a decided and immediate advantage in securing a better view of the parts.

The incision has for its centre the prominent olecranon, extends upwards over the olecranon fossa of the humerus, and downwards along the posterior ridge of the ulna, in extent generally about four inches, and median in position. The incision is carried down to the bone, dividing the triceps aponeurosis and the posterior ligament of the joint.

By a suitable retractor the divided skin and triceps aponeurosis on the inner side are held aside, while the inner surface of the olecranon process and the internal epicondyle are laid bare.

The knife should be kept close to the bone and the periosteum separated as far as possible. If this be faithfully followed the ulnar nerve is drawn inwards with the separated internal lateral ligament and the flexor group of muscles.

The retractor is now transferred to the outer side, and the tissues separated from the olecranon on its outer aspect and from the external epicondyle. Part of the anconeus should be preserved as already indicated.

By flexing the forearm the lower end of the humerus can now be thrust through the wound and its anterior surface cleared. It is then seized with lion forceps and divided at right angles to its long axis. This is attained by holding the humerus in the vertical position. The line of division is just below the epicondyles.

The soft parts should be protected.

The upper end of the ulna and radius are now rendered prominent and made to project through the wound. The attachment of the anterior ligament is detached, the olecranon seized with lion forceps, and the saw so applied as to remove a part of the head of the radius and the olecranon at its base at one division.

The remaining cavity is now irrigated, the tourniquet removed, and the bleeding points secured.

The edges of the triceps should be sutured and a drain used.

In fixing the limb after an excision the forearm should be in a position midway between supination and pronation, and the fingers left free. As to the fixation angle there is some difference of opinion; some suggest an open angle, some a right angle, some less than a right angle. All claim a good result.

Passive motion should be commenced when the wound is healed.

Of the other methods of excision a few words will suffice.

A strictly subperiosteal method is described by Farabeuf. The same incision is employed as that just mentioned, and practically the only difference is an attempt to make the operation more decidedly subperiosteal. Then there is the H-shaped incision of Moreau, consisting of two vertical and a transverse incision, the latter completely dividing the triceps. Some omit the outer vertical limb of the H (Liston), others the inner (Roux).

Instead of a median vertical incision it was recommended by Chassaignac to make the incision along the outer side of the olecranon, and by Langenbeck along the inner side.

The exposure of the joint by lateral incisions is regarded by some as more suitable in cases of ankylosis. In Ollier's method the lateral incisions are of unequal length. That on the outer side commences about 2 to $2\frac{1}{2}$ inches above the external epicondyle, between the triceps and supinator longus, is continued down to the epicondyle, and then obliquely across the olecranon to its inner border, down which it is continued for $1\frac{1}{2}$ to 2 inches. This is the bayonet-incision.

That on the inner side is only about an inch in length, and is made over the internal epicondyle. The bones are denuded of their periosteal, ligamentous, and muscular coverings through these openings, and the operation concluded as in the other method.

In Hueter's modification the lateral incisions are also of unequal length. The inner incision, like Ollier's, is only about an inch in length, and is over the internal epicondyle, but more in its anterior aspect. Through this incision the internal lateral ligament and flexor group are separated. The external incision, 4 inches in length, has for its centre the external epicondyle, is situated more posterior than anterior, and is practically vertical. Through this incision the head of the radius is exposed and the orbicular ligament divided. This enables the head of the radius to be removed. Through both incisions the lower end of the humerus is deprived of its ligamentous connection and periosteal lining, and is removed. The olecranon is divided at its base.

According to Mideledorf the results after excision, as indicated by collected statistics, are

seventy-five good joints, twelve and a half stiff, and twelve and a half flail, in one hundred cases.

Erasion of the Joint.—In tuberculous disease of the elbow-joint in children it is often possible, and whenever possible better, simply to excise the diseased tissue than to perform a classical excision.

The joint is approached by a posterior median incision, or by a transverse incision, and the steps of the operation are similar to those of excision, stopping short at removal of the articular bony ends. The implicated synovial membrane is better cut away than scraped away.

The operation may also be done by two lateral incisions (Watson Cheyne) which are carried down to the capsule of the joint on each side, avoiding the ulnar nerve on the inner and the posterior interosseous nerve at the lower part of the outer incision.

The muscles are to be detached from the condyles, the lateral ligaments divided, and the capsule, freed from its surroundings, cut from its attachments to the bones.

The ends of the bones can now be protruded, examined for any focus of disease, and replaced; the wound then sutured, drained if necessary, and dressed.

Passive movement should be begun when the wounds have healed, as it has been noted that good results as regards movement have been obtained without early movement being practised.

The results of erasion as compared with excision are more satisfactory in respect to subsequent shortening. After erasion there is little or no shortening, and the development of the limb is very slightly if at all impaired.

The same cannot be said after excision when there is necessarily always some shortening varying from two to four inches, with a corresponding want of development in the whole limb. Ankylosis is more frequent after erasion than after excision.

Elder Flowers.—The water of Elder flowers (*Aqua Sambuci*; dose—1 to 2 fl. oz.) is used to flavour medicines; the flowers come from the *Sambucus Niger*, and they contain a resin, a volatile oil, and valerianic acid. *See* Sambuci Flores; Prescribing.

Elecampane.—A composite plant (*Inula Helenium*), known also as the Horse-heal, having bitter aromatic properties, and once used as a tonic; also, a sweetmeat flavoured with the above; the root, by distillation with water, yields *helenin* (C_6H_8O), *inulol* ($C_{10}H_{16}O$), *lactone* or inulic anhydride ($C_{15}H_{20}O_2$), *alantic acid* ($C_{15}H_{22}O_3$), and *alantol* ($C_{20}H_{32}O$).

Electrical Chorea. *See* Chorea (*Electrical*); Dubini's Disease; Hysteria (*Motor Disorders*); Spasm (*Varieties, Paramyoclonus Multiplex, Diagnosis*).

Electricity.

See also Balneology (*Radiation Baths*); Burns and Scalds (*Causes*); Constipation (*Treatment, Massage, and Electricity*); Diabetes Insipidus (*Treatment*); Electrolysis; Eyeball, Injuries of (*Injuries by Strong Light*); Facial Nerve, Paralysis of (*Electrical Reactions of Muscles*); Hydropathy (*Electric Baths in Rheumatoid Arthritis*, etc.); Hysteria (*Treatment, Electric*); Indigestion (*Treatment by Electricity*); Medicine, Forensic (*Death from Lightning and Electric Currents*); Muscles, Diseases of (*Myotonia Congenita* (*Electrical Reactions*); Myasthenia Gravis (*Myasthenic Reaction to Electricity*); Nerves, Multiple Peripheral Neuritis (*Symptoms, Motor, Electrical*); Paralysis (*Family Periodic, Symptoms, Electrical*); Paralysis (*Landry's Paralysis, Symptoms, Electrical Reactions*); Paralysis (*Anterior Poliomyelitis, Symptoms, Reflexes*); Physiology, Tissues (*Muscle, Nerve*); Skin, Pigmentary Affections (*Bronzing*); Spinal Cord, Medical (*General Symptomatology, Electrical Reactions*); Tabes Dorsalis (*Treatment, Electrical*); Tetany (*Symptoms, Motor, Electrical Reactions*); Tumours, Inoperable, Treatment (*Electricity*); Uterus, Non-Malignant Tumours (*Fibroids, Electrolysis*); X-Rays (*High Frequency Currents*).

The value of electricity as an adjunct to the practice of medicine is variously estimated according as it is used for diagnosis and prognosis on the one hand, or for treatment on the other. Most physicians will easily allow the immense aid it may give in the accurate diagnosis and prognosis of certain nervous diseases, while it is comparatively few who are at all enthusiastic as to its beneficial effects in treatment. The fault lies partly in the method of its administration, which is too often placed in incompetent or careless hands, and in no attempt being made to accurately measure the dosage, as would be considered a *sine qua non* were any drug being used. Weak batteries and currents of homœopathic strength are often used, and when no good result accrues "electricity" is said afterwards to have been tried and found wanting. It is, then, of the utmost importance that the greatest care should be exercised in its administration, if an opinion is to be expressed as to its

value in any particular case. The battery, if not in constant use, should be carefully tested first, and a current measurer always used when possible. Now that the constant current is being largely used for electric lighting in houses, the chief source of error due to failure of the battery will be eliminated by its use for medical treatment. The chief use of electricity in diagnosis and in prognosis depends on the fact that when muscles are separated from their trophic centre by a lesion of the motor-cell or nerve-fibre, a peculiar chemical change takes place in the muscle substance owing to which its reactions to the faradic and galvanic currents become altered, and the so-called "reaction of degeneration" is found, which varies in degree according to the severity and acuteness of the lesion. By familiarity with the varying phases of this reaction of degeneration, a surprisingly accurate prognosis may often be given as to the recovery or hopeless paralysis in a given case. By its means we are able to distinguish lesions of nerves from divided tendons in cases of wounds of the limbs, functional paralysis from cases of nerve injury or poliomyelitis, and to give a fairly accurate forecast as to when recovery, if any, is to be expected. Since it is only when the lower motor segment, nerve-cell, or fibre is injured or diseased that there is any alteration in the electrical reactions, no help is afforded by this means in distinguishing a functional hemiplegia from that caused by an organic brain lesion, or an hysterical palsy of the legs from the spastic paraplegia due to a transverse myelitis or other organic cord lesion. For treatment, neuralgias, such as sciatica and lumbago, may often be relieved at once by the constant current, and the most obstinate sciaticas are almost invariably improved by persisting in the treatment, even when accompanied by muscular wasting. Choice of the best form of current for treatment of a given case usually lies between faradism and galvanism, and a usually accepted rule for the treatment of muscles is to use the current to which they react best. For cutaneous effects faradism is the more useful. Putting the difference between the two currents in another way, we may say that galvanism is indicated when quantity of current is required to produce chemical effects, as in electrolysis, or in the direct stimulation of wasting muscle, whereas faradism has more effect in nerve stimulation and in overcoming the resistance of the skin, owing to its much higher voltage than medical galvanic currents.

The forms of electricity in use may be divided into five classes:—

(1) Static or frictional electricity.

(2) High frequency currents, or the oscillatory discharge.

(3) Magneto-electricity, or the interrupted current of an induction coil.

(4) Alternating and sinusoidal currents.

(5) Galvanism, the continuous battery or voltaic current.

Each of these forms of electrical energy are used in medicine in appropriate cases.

Static electricity, often known as franklinisation. This is obtained from the "influence" machine of Wimshurst. Really efficient machines must have large plates, 24 to 30 inches in diameter, and not less than four pairs, more plates giving a proportionately larger amplitude of current, with thicker sparks, while the length of spark depends on the diameter of the plate. These machines may

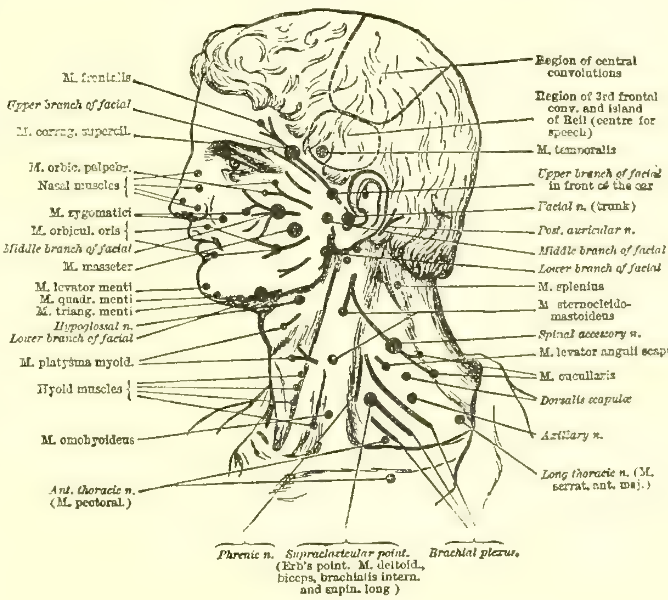


Fig. 1.

also be used to excite a Crookes tube for X-ray work, or, with Leyden jars and properly insulated induction coils, to produce high frequency currents. In treatment, the length of sparks used is regulated by the distance between the discharging knobs, and sparks may be applied to the bare skin, or the electrode rubbed over woollen or flannel clothing. Such treatment is often of great service in dissipating functional symptoms, pains, and hemianæsthesia. No exact physiological data have yet been brought forward to prove its alleged effect on metabolism, an effect scarcely to be expected from the fractional ampèreage of the current. With Leyden jars the shocks become more severe, and a modified form of treatment is Morton's "static induced current," in which the electrodes

applied to the patient are connected to the outer coatings of both condensers, by which means the muscles are thrown into tetanus as by faradism.

High frequency currents may be obtained either from a large induction coil, a Wimshurst-Holtz machine with condensers and coils, or by utilising the alternating current of the street mains. The current produced is of extreme tenuity, but of enormously high potential. The sparks are painful, but if the electrode be grasped in the hand nothing can be felt, though an incandescent lamp will glow if placed in circuit between two persons connected to the

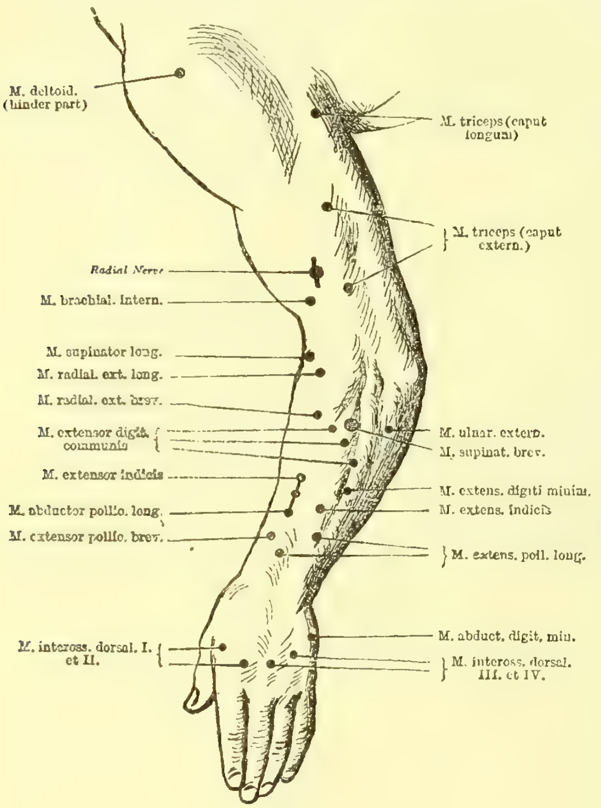


Fig. 2.

terminals. This current, at a pressure of several hundred thousand volts, and oscillating a million or more times a second, if applied to the body by means of electrodes, produces only slight numbness and anæsthesia.

Magneto-Electricity or Faradism.—This current is produced by induction in a long coiled copper wire by suddenly magnetising a soft iron core. Outside this primary coil is a second coil of more numerous turns of fine wire. A usual proportion will be 700 turns of moderately thick wire for the primary coil, and 5000 turns of fine wire in a secondary coil. Two different currents are thus produced, the primary and the secondary. The primary or extra current of the coarse coil is the current that causes most of the sparking of the automatic interruptor. When the soft iron core is pulled out of the primary coil this sparking is much diminished, although the actual battery current is now slightly greater, owing to its having less work to perform in opposing the induced current at make. The secondary current may be regulated in strength either by sliding the secondary coil nearer or farther away from the primary, or by partially withdrawing the soft iron core, or by both means. The primary current is regulated only by pulling out the soft iron core, and the position of the secondary coil does not affect it. The cheaper forms of coil use the soft iron core as the magnet for breaking the battery current, in which case the primary current cannot be varied, but the better class of coils have a separate magnet for the automatic interruptor, which should be a rigid bar working against the tension of a coiled spring to ensure greater smoothness of vibration. Newer types of machine have a separate cell for working the interruptor, in which case the rate of interruption is not affected by the strength of the coil current. The rate of vibration usually varies from 30 to 50 per second, though various devices are used for slowing or increasing the periodicity. Within certain limits the rate of vibration can be controlled by the contact screw of the interruptor. Engelmann's machine, made by Waite and Bartlett of New York, is fitted with a motor interruptor, and the rate of vibrations can be controlled at will from 1 to 800 or 1000 per second, or even more. Wehnelt's electrolytic cell may be used for producing high speeds of interruption, as in X-ray work, but a high voltage is necessary to work it, 24 volts or more. The faradic current is, strictly speaking, an alternating current, but since the make shock is much weaker than that at break, it may be considered as practically unidirectional, and we may speak of a kathode and anode. The kathode is easily found, as it is the pole which elicits the stronger muscular contraction; this is true even in wasting muscle, so long as the muscle responds at all to faradism. There is no electrolytic action in this current.

The faradic current is used for diagnosis as the secondary current, and principally for estimating the irritability of nerves and muscles. It is, however, sometimes used for the diagnosis of nerve pain, as to whether neuralgic or due to neighbouring inflammation, as in Apostoli's method, by introducing one pole into the vagina or uterus in cases of pelvic pain. Neuralgic ovarian pain may often be cured by such an application of the fine coil current, while pain resulting from inflammation is unaltered. In

diagnosis by faradism the unipolar method is most convenient, though sometimes the localised extra condensation of current produced by using the bipolar method, both small electrodes, in close proximity over the nerve or motor point, will elicit a contraction unobtainable by the former method. With the unipolar method the indifferent electrode is large, and placed over the spine or sternum. The active electrode should be small and circular, and fixed on a handle supplied with a spring make and break connection for the finger. The cords connecting the electrodes with the binding posts are best made of twisted fine copper wire covered with silk, ending in stout metal plugs to fit into the screw holes on the battery and electrode. They should be from four to six feet long, and a useful addition is to have the last eighteen inches next the electrode covered with good rubber tubing fitting closely, to prevent the silk getting wet. Cotton-covered wires are objectionable because, even when dry, they allow leakage of current, which is unpleasant both for operator and patient.

The faradic current may be used in diagnosis either as single interrupted shocks, which are produced by a special lever attached to the battery, or by working the vibrating hammer slowly by hand, or else as the rapidly interrupted current. In the former case a single brisk twitch results, and the secondary coil must be shifted nearer to the primary than if rapid interruptions are used. The faradic current stimulates the muscle only through the nerves or nerve-endings, and thus for each muscle there is a certain point, the motor point, where the electrode obtains the maximum contraction, the nearest point to the entry of the nerve into the muscle. These motor points are shown in the accompanying illustrations. When the nerve to a muscle is injured or diseased, the irritability to faradism gradually diminishes and disappears. It is therefore useful in distinguishing a supranuclear from an infranuclear lesion, for if the nerve cell and fibre supplying the muscle are intact, the muscle will respond briskly to faradism, unless there be great wasting from idiopathic muscular atrophy, or following hemiplegia. In these exceptions the muscle gives a weaker and more sluggish contraction both to faradism and galvanism, while in acute myositis the faradic reactions remain intact, unless wasting follows. Therefore in lesions of the upper neuron, such as hemiplegia, spastic paralegia, Friedreich's disease, and all functional paralyses, the faradic reactions are unaltered, and the muscles do not waste, or only slightly. On the other hand, in infantile paralysis, diffuse cervical or lumbar myelitis, peripheral neuritis, and nerve injuries, the faradic reactions will be lost on account of the trophic cell centre of the muscle being separated from it or destroyed. Curarised muscle will, it is true, react to faradism, though badly, but there is not then the profound metabolic change

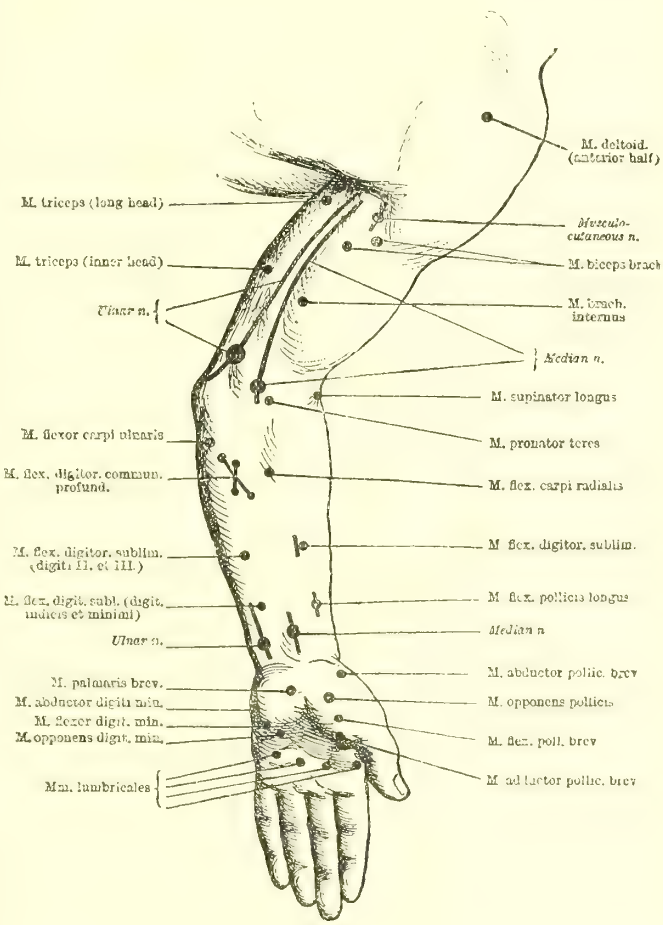


FIG. 3.

produced when the axis-cylinders are destroyed. In all cases in which muscle-testing is being performed, it is of the greatest importance to see that the electrodes are thoroughly wet, the skin well moistened, and also that the indifferent electrode is in good apposition to the skin. A practical point which should always be observed, unless the physician thoroughly knows his battery, is to test the current personally upon the hand before applying it to the patient, so as to avoid giving unnecessarily severe shocks, and also to prove that the battery is in working order.

Treatment by Faradism.—Certain distinctions

are drawn between the action of the currents from the coarse and fine coils respectively, and at slow or rapid interruptions. The two currents differ in their electromotive force and in volume of current, that of the primary or short coarse coil having lower voltage and greater volume, that of the secondary or long fine coil having high voltage and less actual ampèreage. The current from the long fine coil will then be specially adapted for high resistances, when localised sensory effects are desired, as in treatment of the dry skin with the wire brush. The primary coil will be more useful for low resistances and diffuse muscular effects, as in treatment of constipation, with one pole in the rectum, and a large moist abdominal electrode. With wet electrodes and well-moistened skin the fine coil current has powerful effects on muscle, and the physiological effects, both muscular and sensory, increase with the rapidity of interruption up to 50 or 60 per sec., which is about the maximum of interruption obtained with an ordinary well-constructed instrument. Hence, in practice, the primary coil is rarely used by most practitioners, both muscular and sensory effects being obtained from the long fine coil.

The best machines have the outer movable coil made of three different thicknesses of wire, which can be tapped at various lengths, or the whole used if necessary. Generally speaking, fine coil currents, with rapid interruptions, are indicated for the treatment of anæsthesia and neuralgic pain, while coarse coil currents are better for producing muscular effects with low resistance, as by internal application. The most useful speed of interruption is 40 or 50 per sec. With slower speeds and well-moistened skin there is less pain produced and good muscular contractions, tetanus becoming resolved into single contractions if slower than 10 or 12 per sec. With high speeds of interruption, 300 per sec. and upwards, the current is not felt, and no muscular effects are produced, slight numbing of the skin only.

Faradism is useful in the later recovering stage of muscular wasting, and in organic nervous diseases is often of use in improving anæsthesia, and also in keeping up the nutrition of skin and muscles, reddening of the skin always following its application. It is doubtful whether the current has any curative effects upon the nerves or cord.

In neurasthenia and hysterical paralyses and anæsthesias, faradism is of great service, and may be applied locally or generally by the bath treatment. The *faradic douche* may be applied to the spine and limbs by combining faradism with douche treatment, one electrode being applied to the patient's chest, and the wire of the other fastened to the metal douche or syringe. Severer treatment is used in hysteria by means of the wire brush for one electrode, which is applied to the *dry* skin, and dabbed, not stroked, upon it. This is exceedingly painful, and is sometimes used in arousing patients found comatose from drink or morphia. In dangerous chloroform narcosis it is also used as a stimulant, and the phrenic nerves may be stimulated, though this is of doubtful benefit. If the wire brush be used for any length of time it may cause an irritable pimply rash to appear, and sometimes even bleeding points.

In the treatment of neuralgic pain faradism is often of great service, either alone or combined with galvanism. Severe neuralgic headache may often be greatly relieved by faradism applied to the forehead and back of the neck for ten minutes at a time. It is very soothing, and may induce sleep. Combined with galvanism it is also of use in treating sciatica, when galvanism alone fails to give marked relief.

Galvanism.—The facilities afforded by the use of the constant current for electric lighting are being now extensively used for the application of galvanism in medical practice. The street currents are of varying pressures, from 100 volts to 250 volts in different districts, the Board of Trade maximum for low pressure currents having been recently raised to 500 volts. The main current may be used to charge accumulators when a portable battery is needed, or for hospital or consulting room use it may be led direct on to a switch-board, such as that devised by Milne Murray, where it can be used to drive an induction coil, for cautery, or for application as galvanism. For this latter purpose especially it is necessary to reduce the voltage, which is first done by placing an eight-candle lamp in the circuit as a safety resistance. It is then further reduced by passing the current through a graduated rheostat, usually made of graphite. It is found, however, in practice that patients treated thus in series with the dynamo complain of a burning sensation, even when the galvanometer is showing less current than they could easily bear from a Leclanché battery. This is probably due to rapidly varying voltage, and can be obviated by arranging the rheostat so that the patient is not in series with the dynamo, but on a shunt. In Milne Murray's apparatus the rheostat is made of a long coil of platinoid wire, containing no platinum, but having a high resistance. By means of a sliding contact an increasing length of this wire is thrown into the alternative circuit to the patient, thus increasing the amount of current passing through the latter. This is of course more wasteful of current, but the cost is really negligible. Main currents are much cheaper than battery currents, much more constant as a rule, and there are no parts to replace or get out of order. There are a few dangers to note in the use of strong currents from a dynamo; sudden breaks of current at the generating

station are not infrequent, but usually take place at certain times, as when a fresh battery of accumulators is switched on, or the wire of the rheostat might break between the zero point and the sliding spring of the shunt circuit, though this is very unlikely. Any sudden break of the current due to either of these causes would give an uncomfortable shock with a current of 20 m.a., while with the heavy current, such as is used by Apostoli for the electrolysis of fibroids, the consequences might be serious.

Next to using the main current or an accumulator, the best generator for medical purposes is a battery of Leclanché cells, of which from forty to sixty cells in series will yield sufficient voltage for all purposes. For a stationary battery these should be of three-pint size, and may be stored in a cupboard or cellar. The smaller the cell the less the efficiency, but for a portable battery small cells are a necessity. Dry cells of the Hellesen-Obach type are then the most useful, as a sufficient number of small wet cells makes too great a weight. They require no attention, and can be placed in any position, but they wear out in about two years, and must be replaced by new ones. In working with batteries it is of the greatest importance to keep all metal contacts clean and screws tightly fixed, and care must be taken to avoid any short circuiting. Sometimes if a battery of wet cells is neglected, one or two cells may become short circuited, as by the zinc rod resting against the carbon. This will soon increase enormously the internal resistance of the cell, and diminish the strength of the battery. Such a cell may be recognised by a brown discoloration appearing at the surface of the liquid, which soon becomes precipitated on the surface of the glass jar. Usually the zincs require scraping, and the liquid in the cell to be filled up every six or twelve months. A galvanic battery, to be efficient for diagnosis and treatment, requires, in addition to a current collector, a reliable galvanometer and a pole commutator or current reverser. The best galvanometer for a stationary battery is Edelmann's large horizonal one, costing £10, while for a portable battery Schulmeister's floating galvanometer is the best, costing from £2 : 10s. to £3. All galvanometers for medical work are graduated in milliampères, and should be provided with a screw shunt to shut off $\frac{9}{10}$ths of the current from the instrument, thus increasing tenfold its working limit. The current reverser is necessary for diagnosis, while for treatment it is useful to have in addition an automatic current interruptor or reverser, breaking or reversing the current once or twice a second. This can be arranged either by clockwork, or by a separate electro-magnet and spring hammer. Most good batteries are combined to give faradism or galvanism, or both together, from the same pair of binding screws. This is effected by means of De Watteville's switch. Such a battery with galvanometer and accessories is priced in Schall's list at from about £12 to £18, according to the number of cells. A simple galvanic battery of twenty-four to thirty-two cells for treatment merely costs from three to four guineas. Small faradic batteries are much cheaper, and can be obtained for 10s. and upwards.

A good battery should also have available to place in circuit a metal rheostat graduated to at least 1000 ohms, by the use of which, and a

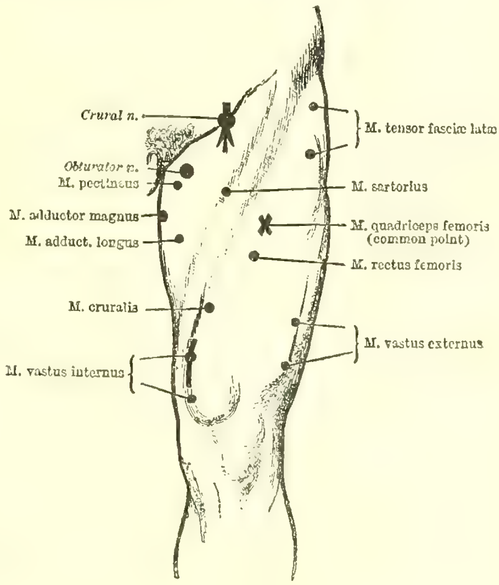


Fig. 4.

double current collector, not only can the voltage of the battery be tested from time to time, but the E.M.F. of each cell can be ascertained, and a faulty cell thus located.

A practical point to remember in treating with galvanism, as with faradism, is to keep the electrodes well soaked and the skin thoroughly moistened, for the amount of current depends as much on the resistance of the circuit as on the voltage, or number of cells used. The skin offers by far the larger part of the total resistance, and to make sure of good contact, it is a good plan to place, if convenient, one limb with the indifferent electrode in water.

The galvanic current, though perceptible to the sensory nerves during the whole period of its flow, yet, unlike the faradic, stimulates the muscles to contract only at the closure of the circuit, so long as the strength of the current does not vary suddenly during its application. This is true so long as moderate currents only are used, but with powerful currents tetanus may be produced. In normal muscle a quick twitch is produced at make, owing to the stimulation of the nerve-endings, and, like the faradic, may be used to stimulate the nerve at a distance

from the muscle, or the nerve-endings in the muscle by direct application to the skin over the motor point. No contraction is produced at break except with currents too powerful to be borne. The motor point then holds good for both faradism and galvanism in normal muscle, but when the nerve-endings are destroyed, though the faradic current is inert, yet galvanism is still able to cause the muscle to contract when applied directly over the part, though the contraction is no longer a brisk twitch, but sluggish and prolonged. This is the essential feature of the so-called *Reaction of Degeneration*, or R.D. This change is more marked the acuter the nerve lesion, and affects the irritability of both

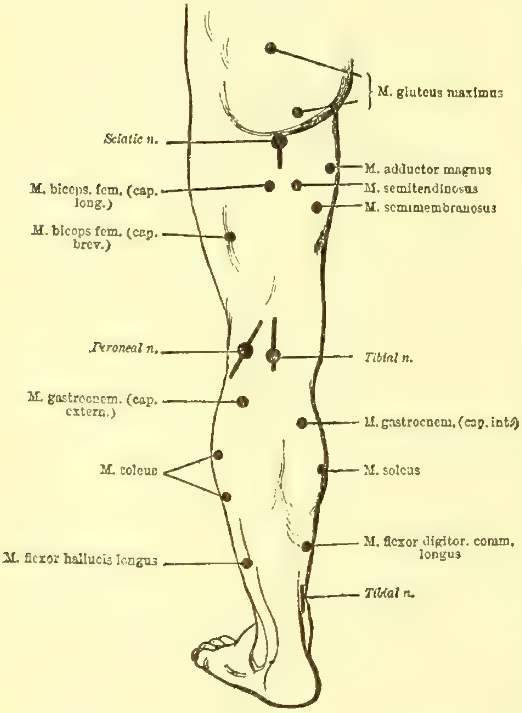


FIG. 5.

nerve and muscle. In a typical case there is in the *nerve* a transient wave of hyperexcitability to both currents below the lesion, followed by a gradual diminution to both currents without polar change, until, in a severe case, there is complete loss of reaction in the nerve at the end of a week or ten days. In the *muscles* the faradic reactions also steadily diminish to complete loss after the first week, while the galvanic irritability, at first slightly diminished, soon rises during the second or third week above the normal, though the contraction is sluggish, with usually a marked polar change, ACC $>$ KCC. In the early stage there may be a quick twitch with KCC and a sluggish contraction with ACC. The opening contractions also become more easily obtainable, and tetanic contractions are seen at make with the kathode and even with the anode. This condition may last several weeks to two months. If the nerve does not recover the faradic reactions never reappear, while the galvanic irritability also diminishes, in spite of treatment, and eventually no contractions will take place to either current. This takes many months or even years to happen, and during the later stages the kathodal contractions usually reassert their supremacy, or KCC may = ACC. Erb has described a partial form of R.D. which is met with in less severe nerve lesions, and is therefore of better prognosis. It consists of slightly lowered irritability of the *nerve* to faradism and galvanism, with diminished *muscular* irritability to faradism and hyperexcitability to galvanism, with the polar change ACC $>$ KCC. This may be apparent without any loss of voluntary power, and is often to be met with in cases of nerve injury in muscles in the neighbourhood of those paralysed, *e.g.* the deltoids may show partial R.D. in cases of wrist-drop from lead neuritis, without any obvious weakness at the shoulders. Sometimes, too, as in facial palsy following shortly after mastoid operations, the voluntary power may be completely lost for a short time, yet the only change is diminished irritability to both currents in nerve and muscles, with no galvanic hyperexcitability or polar change. Such electrical change is of better prognosis than when full R.D. is present, and some return of voluntary power is usually to be seen towards the end of the second week. Lastly, in recovery from severe paralysis, such as peripheral neuritis or injury, considerable voluntary power may return before any faradic irritability, and full voluntary power may be re-established with diminished faradic and galvanic excitability remaining for several months.

In practical muscle-testing it is important to place the indifferent electrode at a distance, as on the spine or another limb. The first few contractions obtained should be neglected, since the muscles often require some stimulation before giving their best contractions. The strength of contraction on the two sides should be compared, and in testing or treating the small thumb muscles it is necessary to use twice the number of cells required to send the same current through the muscles of the forearm, owing to the thickness and great resistance of the skin of the palm.

Treatment by galvanism is indicated in cases of muscular wasting from nerve injury, peripheral neuritis, or spinal disease, in which the faradic reactions are diminished or lost. The electrodes should be well covered with flannel or chamois leather, as electrolysis is set up in the skin with metal contacts. The kathode or negative pole is the more painful, especially if allowed to get at all dry. This pole is usually selected for treatment, but either pole may be used according to the strength of contractions

each produces. The moistened electrode should be stroked over the skin of the affected part, or, if two limbs require treatment, each may be placed with one electrode in a separate pan of warm water, and the current reversed once or twice a second; rapidly-interrupted galvanism produces no contractions when R.D. is established. This is very conveniently done by an automatic current reverser attached to the battery, and the method is especially suitable for young children with infantile paralysis, as, placed with their backs to the operator and the battery, they are much less frightened, the current being turned on cell by cell, with intermittent reversals. The strength of current required will vary from 5 to 15 milliampères, and even quite young children will bear reversals of 10 milliampères if given by this method. In testing children satisfactory results are often difficult to get without using an anæsthetic. On the face much weaker currents must be used than on the limbs; 3 m.a. is usually all that can be borne at first, which may be increased to 5 later. On account of the sensitiveness of the skin of the face the anode may be used, as it causes less pain. Galvanism to the face and head are also objectionable on account of the flashes of light in the eyes and the unpleasant metallic taste produced in the mouth. The use of the anode is, however, useful in trigeminal neuralgia with painful, tender spots. Care should be taken in treating the face or head not to reverse or suddenly interrupt the current when currents of more than 10 m.a. are being used, as the sudden shock might be dangerous.

In treating sciatica the patient should sit on one moist pad, usually the anode, and the other electrode is best put in a pan of warm water, in which the foot is placed. The current is then gradually turned on to give at least 20 milliampères for ten or fifteen minutes. Some patients can bear 30 to 40 milliampères, or even more. The current must be gradually reduced at the end of the sitting to avoid a shock, and the patient should lift the foot out of the water slowly, leaving the heel last. The choice of pole makes little difference, but the anode should be placed on the more sensitive skin. Owing to the diffusion of current in all directions there is no special virtue in so-called ascending or descending currents. Sciatica is almost invariably relieved for some hours by this treatment, and cure is fairly common, even in cases which have resisted treatment by rest and drugs for months. In severe cases combined faradism and galvanism may be used with advantage. Galvanism is best applied daily, and the current should be as strong as the patient can conveniently bear. Small batteries are worse than useless.

In the treatment of peripheral neuritis, on account of the inflamed nerves, galvanism should not be used until the tenderness has mostly disappeared.

Prognosis cannot be attempted until there has been time for the development of R.D. in the nerve and muscle. Considerable paralysis may be present, with only diminished reactions to faradism and galvanism in both nerve and muscle; such cases usually show, under treatment, commencing return of power by the tenth day, and almost complete recovery at the end of a month. If, at the end of the first week, there is no faradic or galvanic reaction in the nerve, with R.D. in the muscle, the case will be at least three months in recovering perfectly. If no faradic irritability appears in nerve or muscle

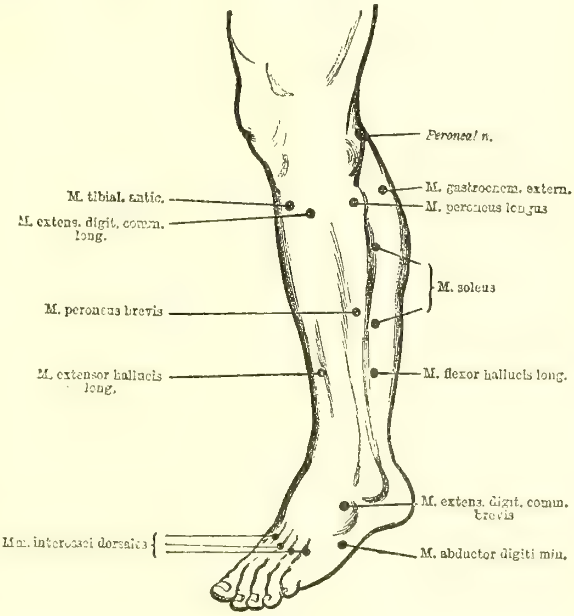


Fig. 6.

after three weeks the case is very severe, and will take six months or a year to recover. If, in spite of treatment, no faradic reaction appears after two months, recovery is hopeless.

Marked improvement sometimes follows in severe cases of nerve injury or facial palsy which have been untreated and *in statu quo* for months, in which persistent treatment by galvanism has been started late. Indications of improvement will be seen in the contractions becoming stronger and brisker, and finally return of the faradic reactions. Such cases are, however, very liable to develop permanent contracture which will not yield to treatment. It is difficult to resist the belief that galvanism has some curative effect on the nerve as well as in maintaining muscular nutrition.

Some of the severest cases of muscular wasting of the forearm are due to the too tight application of splints in fractures or sprains, causing severe diffuse neuritis, with muscular

wasting, glossy skin, and joint adhesions. Occasionally an ischæmic myositis results from this pressure, in which, though the reaction of degeneration is found, there is primary and early muscular contracture.

In *gynæcology* the method of electrolysis of fibroids was brought forward in 1882 by Apostoli as a substitute for operative treatment. The principle of treatment is the introduction of a platinum sound into the cavity of the uterus, which is connected with the positive pole of a powerful galvanic battery. The negative electrode used is made of moist modeller's clay, applied to the abdomen, on it being placed a metallic contact connected with the zinc or positive element of the battery. The current must be turned on gradually after everything is in position, and great care must be taken to avoid giving shocks, owing to the powerful currents used. At the first sitting never more than 50 m.a. are given for five minutes, and if this is followed by increased pain or inflammatory reaction the treatment is considered contra-indicated. The treatment may be carried out two or three times a week, increasing the strength of current to 100, or even to 200 milliampères. The results claimed are diminution in size of the fibroid, and cure of hæmorrhage and of the feeling of discomfort. Apostoli advocates the preliminary use of intra-uterine faradisation with the fine coil or the sinusoidal current for diagnosis, when the treatment is contra-indicated if the pain is aggravated. Interstitial and submucous fibroids, subinvolution of the uterus, chronic parametritis, are the most suitable for electrolysis; acute uterine or periuterine lesions, suppurative parametritis or salpingitis, and cystic tumours of ovaries or tubes, contra-indicate the treatment. Continued cauterisation with the positive pole of the lining membrane of the uterus is said to lead occasionally to atresia of the cervix. This may be obviated by once or twice using the negative instead of the positive electrode for intra-uterine use.

This effect of the negative pole in dilating a stricture has been used for strictures of the urethra, rectum, œsophagus, lachrymal punctum, and nasal duct. Weak currents only should be used, from 5 to 10 m.a.

Electrolysis is also used, as in the treatment of nævi, for destroying hair follicles on objectionable moles, growths of hair on the cheek or chin in women, and for inverted eyelashes or trichiasis.

The kathode is also used by some ophthalmic surgeons for absorbing old corneal leucomata, 2 m.a. being used for three minutes at a time, and the constant current also for treating persistent blepharospasm following keratitis, anode to back of neck, and kathode over the closed lids. In optic atrophy galvanism has been said to improve vision when consecutive to neuritis, though apparently useless in primary optic atrophy. Paralysis of ocular muscles may also be treated by galvanism, the physician's finger making a convenient electrode.

Sinusoidal Current.—The use of the alternating current for electric lighting provides a ready means of using in medical treatment the sinusoidal current, so called on account of the regular periodicity of the curve representing the rise and fall of electromotive force. The alternating current is supplied from the dynamo to the street mains at the high pressure of 2000 to 5000 volts, alternating 80 to 110 times a second in this country. By passing a current through a transformer made like an induction coil, an induced current is generated in the outer coil, which is made of coarse wire when intended for the low resistances of cautery or lamp, and of fine wire for the high resistances of medical treatment. No automatic interruptor is required on account of the current in the primary coil being already alternating. With a suitably made transformer the outer coil may be tapped at varying lengths, and a current of any required voltage obtained, up to 100 volts, at which pressure the current is brought into the building. The sinusoidal current is much smoother than the faradic, though its effects on nerve and muscle are somewhat similar. Muscular contractions and tetanus are produced, and a certain amount of numbing and sedative effect after a certain time. The best effects are seen in bath treatment, the forearms or feet being placed in two separate pans of warm water with an electrode dipping into each, the current being applied for ten minutes. Doulton's 3-gallon tongue pans (3 shgs. each) serve admirably. For general application the patient is immersed in a warm bath, well enamelled inside, and flat electrodes to the spine and feet.

Broadly speaking, the indications for using the sinusoidal current are the same as for the faradic. It is a smoother, less jerky current, has more pronounced sedative effect, while the muscular contractions are somewhat less vigorous.

The Electric Bath.—Bath treatment is the most convenient and pleasant form of administering either galvanism, faradism, or the sinusoidal current to the whole body as a general tonic or stimulant in neurasthenia, peripheral neuritis, anæmia, debility, rheumatism, and defective nutrition generally. A well-enamelled metal bath suffices fairly well, but an oaken or porcelain one insulates better. If the continuous main current is to be employed the water taps should not be attached, but should overhang the bath, and the waste pipe should be quite short and discharge into an open gully, so as to insulate the bath from earth contact, to prevent leakage of current. Large zinc or copper electrodes are used, the anode placed behind the back and protected by a back-rest of

cane or webbing, and the kathode placed close to the soles of the feet. Bath treatment is wasteful of current, only one-eighth of the current passing through the patient. The water should be warm, 99° or 100° F.

The faradic current is most useful for neurasthenic pains, and the coarse coil or primary current may be used, but the interruptions should be rapid. For rheumatism, chlorosis, and general metabolic effects, the continuous or battery current is more likely to be successful. It may, however, cause throbbing of the head and faintness when strong currents are being used. Since seven-eighths of the current are wasted in the water, the galvanometer in the circuit must indicate from 150 to 200 m.a. to ensure a current of 20 m.a. passing through the patient. The current should be turned on gradually, and care taken to avoid shock.

Dangers of High Tension Currents.—Currents at pressure over 500 volts are known as high tension, and are very dangerous to handle. Accidents are not liable to happen inside buildings, because the voltage is always reduced to safe limits, but in electric lighting stations, in the operation of changing transformers, and in working with overhead wires, a sudden shock at 2000 or 5000 volts pressure often proves fatal through syncope, and bad burns often result, even if there is recovery. Accidental contact with low pressure of 100 or 200 volts is not dangerous, though extremely awkward shocks may be given. In treating patients from the main, a safety resistance should always be included in the circuit, such as an eight-candle lamp, which will not allow more than 300 milliampères to pass.

It is not unusual to hear the continuous current spoken of as the low tension, and the alternating as the high tension current. A current of 100 volts pressure and 1000 ampères volume gives the same number of kilowatts or electric horse power as a current of 10 ampères at 10,000 volts. Either current may be produced at high tension, depending on the construction of the dynamo, and recently some electric light installations have been built for high tension continuous current, as that at Oxford, which supplies current of 1000 volts pressure. This voltage is too high for direct supply to the consumer, and therefore transformers have to be used to lower the voltage to 200 or 100 volts. This is performed by motor rotary transformers, built as combined motor and dynamo, differing from the stationary transformers of the alternating current supply. In this country all cables are laid underground, with the exception of some tramway and railway lines, but in America and on the Continent overhead wires, even for high tension currents, are not uncommon, increasing the risk of accidents from collapse of buildings or supports.

The continuous current is often supplied to the mains on the three-wire system, two outer and one inner wires. Workmen sometimes get severe shocks by touching the two "outers," getting a "short," or short circuit. Up to 250 volts, after the momentary shock there may be faintness, pallor, haziness of vision, tremor, and loss of nerve for a day or two. With higher pressures of 2000 volts and upwards death from failure of heart or respiration may be instantaneous, and this fact has been made use of in America for the electrocution of criminals. Death is not a constant result, however, and though severe convulsions are produced at the time of contact, the patient may ultimately recover with no worse result than a bad burn.

Experimental results on dogs by Prevost and Battelli with alternating currents showed death from heart failure with shocks at 120 volts pressure, but with high tension currents of 1200, 2500, and 4800 volts, tetanus and convulsions were produced, and respiratory failure, but the animals could be saved by artificial respiration.

In view of these results, and the occasional recovery of human beings after shocks at these high pressures, it would be extremely desirable in all cases of apparent death from electric shock or lightning stroke to make use of artificial respiration at the earliest possible moment as a desperate remedy.

Electro-.—In compound words *electro-* (Gr. ἤλεκτρον, amber) has the meaning of "relating to electricity." *Electro-bioscopy*, for instance, means the examination of a body by electricity in order to ascertain if life be extinct (viz. by absence of muscular contractions); *electro-anæsthesia* is the loss of the electric sensations (*vide* HYSTERIA); *electro-biology* is the science of animal electricity, of electricity in living bodies, or, in a special sense, "animal magnetism" or hypnotism; the *electro-cautery* is the galvanic cautery (*vide* GALVANIC CAUTERY); *electrocution* (*electrical execution*) is the use of the electric current for the execution of criminals, as employed in the United States of America; an *electrode* is one of the poles of a galvanic battery, or the terminal of the open electric circuit; *electro-diagnosis* is the use of electricity for the purposes of the recognition and distinction of diseases; *electro-dentistry* is the employment of electricity in dentistry (*e.g.* electro-cataphoresis, *vide* TEETH); *electro-endoscopy* is endoscopy carried out with the aid of the electric light; *electro-hæmostasis* is arrest of bleeding by the electric current; *electro-massage* is kneading a part (*e.g.* a limb) with a roller and a small galvanic machine; *electro-medication* is the use of electricity for medical purposes, and, in a special sense, the introduction of medicines into the system by means of electricity; *electro-pathology* is the investigation of morbid conditions by the testing of the electrical reactions.

of muscles and nerves; and *electro-puncture* is the electrolysis of a part of the body or of its tissues by means of a needle electrode (this method of treating uterine fibroids is now generally abandoned).

Electrolysis.

See also Aneurysm (*Treatment, Electrolysis*); Capillaries, Diseases of (*Treatment of Angioma, congenital and non-congenital*); Lymphatic System, Physiology and Pathology (*Dilatation of Lymphatics*).

Electrolysis is the decomposition into simpler elements of a substance placed between the poles of an electric battery. The process as applied to the human body is somewhat complicated. There is first the primary electrolysis, whereby the tissues are decomposed and oxygen and acids appear at the positive, hydrogen and alkalies at the negative pole. But, secondly, these nascent substances necessarily react on anything in their neighbourhood, whether electrode or tissue, with which they have chemical affinity. This secondary electrolysis gives character to the sloughs produced, and these results may be still further modified by the metal of the electrode. Yet, again, the passage of a galvanic current through tissue must undoubtedly have an effect not only at the electrodes where the destruction is produced, but throughout the whole electrolyte. In nerve tissue it may profoundly affect the power of transmitting impressions even up to complete abrogation, and similarly with cells or fibres it may modify or annul their contractility or nutritive properties.

These effects of the constant current of electricity may be applied with beneficial result in various forms of disease, such as angioma, aneurysm, various forms of solid or cystic tumour, stricture, abnormalities of hair, etc.

1. In *angioma* the action is chiefly upon the solid constituents of the growth. At the negative pole the slough produced is diffuse, large, very slightly contractile; at the positive limited, small, and tending to condense in healing; the one corresponding to an alkaline, the other to an acid cauterisation. Practically in electrolysing a nævus we introduce so much caustic potash or nitric acid made out of the tissues. We introduce these nascent caustics in such infinitely minute division, and we so destroy tissue in producing them, that they cannot be carried injuriously into the circulation. By doing this subcutaneously we avoid the evils of putrefaction and we conserve the skin. If properly performed the effect is that after a little tenderness the slough is slowly absorbed without suppuration and without scar.

The cases of nævus, therefore, in which electrolysis should be used are the mixed and subcutaneous varieties on the face or hands, where a scar is to be avoided, and those elsewhere which from size or situation are not amenable to excision. In these cases its advantages are, that it is absolutely safe, that with sufficient perseverance it cannot fail to cure, and that it does so without leaving the slightest mark.

It is necessary to recognise its limitations. It cannot cure a pure cutaneous nævus, or port-wine stain, without leaving an unsightly scar. In the mixed variety the discoloration generally disappears when the subcutaneous portion is destroyed, but in a few, perhaps 2 or 3 per cent, a slight stain remains. Some cases may be cured by a single operation, many require more, even up to 8 or 10, so that when a scar is not important excision is better because quicker.

The method of operating is simple, but requires attention to detail.

The best strength of current is from twenty to forty milliampères. In the neighbourhood of large nerves it should be less. It should never exceed eighty. It should be so arranged that it can be increased from zero without shock.

The needles should be insulated with vulcanite. I know of no improvement on those originally introduced. The length of the exposed point must correspond to the size of the nævus, varying from an eighth to three-quarters of an inch. For the sake of sharpness steel is the best material, but in that case the positive pole requires to be resharpened after each operation.

The positive pole is placed in a portion of the tumour where it can do no harm. It does little though useful and firm work, and need not be moved more than once or twice during an operation. The negative, on the contrary, is held in the hand and constantly moved about. Its action is diffuse and therefore more valuable, but it requires constant watching lest it destroy the skin. This is the only difficulty in the operation—to produce sufficient destruction of the nævus without touching the skin. In early operations it is better to do too little than too much. The amount of swelling and induration are your guides, and the slightest pallor of skin should make you stop.

I find it rarely useful to repeat the operation under six weeks. Very little may start the process of involution, and the destroyed portion is not absorbed under that time.

What has been said of electrolysis in nævus applies with little modification to other forms of angioma. The degree of resistance will vary according to the nature of the tumour. Cirsoid aneurysm and aneurysm by anastomosis are often spoken of as if they were one disease. The application of the names is unimportant, but it ought to be definitely understood that these are two essentially distinct forms of tumour.

The former is a dilatation of the branches of an artery, most commonly the temporal, has nothing to do with capillaries, and is singularly amenable to electrolysis. The latter is a cavernous metamorphosis of capillaries, in which great caverns communicate backwards with enlarged arteries, forward with dilated veins, and which more than any other angioma resists cure by electrolysis. An artery if occluded remains so, and its occlusion often involves the atrophy of a considerable extent of the tumour. In short, I have never failed to cure by electrolysis any cirsoid aneurysm, and several have required only one operation. On the other hand, a vein easily reproduces its lumen, and great caverns and dilated veins are not readily obliterated whether by action on their walls or on the contained blood. I have given up several aneurysms by anastomosis in despair during my early experience. Other forms of angioma may all with patience be cured by electrolysis.

2. Aneurysm.—There is a class of aneurysm, viz. the arterio-venous, which has in some respects an affinity to the aneurysm by anastomosis of which we have been speaking. The murmur, the close association of arteries and veins, and the singular changes in the latter, are very similar in them both. But there is this important distinction, that the arterio-venous aneurysm being usually traumatic, has a comparatively small arterial orifice, and that whether it be an aneurysmal varix or a varicose aneurysm. This renders it likely that arterio-venous aneurysm may be satisfactorily treated by electrolysis. My experience is limited to those on the smaller vessels, but all were successful. The proper course is to work about with a stout negative needle until you have found a place where by transfixion or lateral pressure you can more or less completely control the pulsation, and there maintain it till the electrolysis of the vessel is complete. It appears to me the best form of treatment in these difficult cases. Other forms of aneurysm in which electrolysis is worthy of consideration are those which some surgeons have treated by injection of coagulants. Here we must take into account the effect of electrolysis upon blood. At the positive pole there is produced a small, black, firm coagulum, at the negative a considerable quantity of a loose, pinkish, white froth, and in addition to these a certain amount of thin, dark fluid consisting of serum and altered corpuscles. Comparing electrolysis with other coagulants it is evident that the fluid and frothy products will be prone, the first to disappear, the other to contract, but on the other hand there is no risk of deleterious substances being carried into the circulation, while by bringing the needles for short periods in contact with the walls that inflammatory action may be set up which Mr. M'Ewan aims at in his mode of treatment by needling. Of surgical aneurysms the gluteal and sciatic are those which most naturally lend themselves to treatment by coagulants, and in which I should prefer electrolysis as safest and best.

But the aneurysms which have been most frequently subjected to electrolysis are those like the aortic or innominate in which no ligature or other operation is practicable. More than thirty years ago, in studying this question, I came to the conclusion that the dangers of electrolysis lay in hæmorrhage which came from cauterisation of the needle-track, and in inflammation of the sac which was caused by cauterisation of the wall. The introduction of the vulcanite coating abolished these dangers. Electrolysis of aortic aneurysm at once became free from risk. But it was found that a large part of its beneficial effect had also disappeared, that as a coagulant pure and simple the electric current was not very efficient, and that a large part of the old cures and improvements had really been due to the inflammation produced by uninsulated needles, which was also the source of danger. The problem, therefore, now is to attain that amount of irritation of the sac which will cause coagulation, but not severe inflammation. Short operations with very fine uncoated needles may attain our object in some cases, in others longer operations with insulated needles, whose exposed points shall for short periods and at different parts be made to touch the wall. The comparison in aortic aneurysm lies not with coagulants which are altogether out of court, nor I think with the introduction of foreign material like wire which must remain permanently, but with the plan of prolonged scratching with fine needles. Which is safer and more certain is not yet determined.

When in other aneurysms electrolysis comes into competition with ligature, incision, excision, and compression, the relative certainty and safety must be determined for each case. If I were asked to put the matter generally, I should say that where practicable compression is our first resource, and that for such cases as the popliteal aneurysm ligature comes next, but that the safety of electrolysis brings it in after these notwithstanding its uncertainty.

3. The other applications of electrolysis, as a depilatory (where it is very useful), in the treatment of stricture (where its advantage is very doubtful), and in the destruction of tumours, may be referred to their respective headings. Under the last head, however, I would mention one or two points which may probably otherwise escape consideration. I assume in the first place that, like all escharotics, it may be abandoned in malignant disease. In innocent tumours generally its value may be determined not only *a priori*, but also from experience in uterine fibromyoma. I suppose one may say that electrolysis tends to arrest but rarely succeeds in curing fibroids of the uterus, and that its action is slow and necessitates frequent repetition. It is

therefore to those tumours elsewhere, in which we may legitimately be satisfied with such a process and result, that electrolysis is applicable. For example, I think it possible that electrolysis has a future in connection with soft goitre and similar conditions. In goitre there is a structure to a certain extent comparable to angioma. A good deal may therefore be done by destruction and absorption of the slow-growing tissue. It is not unlikely, moreover, that as in uterine fibroids more than the escharotic action may come into play, and that we may have nutritional and nerve effects from strong currents. My own experience of the local results has been distinctly favourable, but it is right that I should add that I never give an anæsthetic in exophthalmic goitre without fear and trembling, and that patients will not submit to a strong current without it. This risk, however, is involved in all surgical treatment of goitre.

Electron.—According to the views on chemical compounds suggested by the discoveries regarding radium, the atom is not single and indivisible, but is made up of electrons, or "corpuscles," each containing a very small charge of negative electricity; the Beta rays of radium are electrons; perhaps electrons are the *atoms of electricity.*

Electro-therapeutics.—The treatment of disease by electricity. *See* Electricity; Electrolysis; etc.

Electro-thermic Angiotribe.—A form of compression-cautery, introduced by Downes of Philadelphia for the treatment of the pedicle (*e.g.* in ovariotomy).

Electrotonus.—The altered state of excitability of a muscle or nerve while an electric current is flowing through it; there is increased excitability round the cathode and diminished excitability round the anode. *See* Anelectrotonus; Cathelectrotonus; Physiology, Tissues (*Muscle and Nerve, Electrotonus*).

Electrozone.—An antiseptic preparation, consisting of sea-water in which the alkaline chlorides have been converted, by electrolysis, into alkaline hypochlorites.

Electuaries.—A confection, conserve, or paste, consisting of a medicinal substance (usually a powder) mixed with honey or syrup. *See* Confectio; Prescribing.

Eleidin.—A substance allied to keratin, found in the rete mucosum of the skin. *See* Physiology, Tissues (*Epithelium, Keratin*).

Elemi.—An exudation (aromatic, yellowish-white in colour, soft in consistence) from the "pitch-tree" of the Philippine Islands; it contains a volatile oil, resins (*Amyrin*), *amyric acid*, and crystalline bodies (*Bryoidin, Breidin*); it is used for making plasters and ointments.

Elephantiasis. *See* Filariasis (*Pathogenic Effects, Lymph Scrotum, Lymphangitis, Elephantiasis Arabum, Elephantoid Fever*); Leprosy (*Elephantiasis Græcorum*); Mycetoma (*Diagnosis*); Pregnancy, Intra-Uterine Diseases (*Congenital Elephantiasis*); Scrotum and Testicle, Diseases (*Elephantiasis Scroti, Sterility due to Elephantiasis of Scrotum*); Tumours (*Connective Tissue Type, Neurofibromatosis* (*Elephantiasis Neuromatosa*)).

Elevator.—An instrument for raising a piece of bone, *e.g.* in a depressed fracture of the cranium (*bone elevator*), or for keeping a part (*e.g.* the eyelid) out of the way during an operation, or for correcting a displacement of the uterus (*uterine elevator*).

Eleventh Nerve. *See* Physiology, Nervous System (*Cranial Nerves, Spinal Accessory*).

Eliminant.—A medicine or medical means for throwing off some noxious product through the excretions. *See* Diaphoretics; Diuretics; Purgatives; etc.

Elixir.—In modern use an elixir is a liquid containing alcohol, syrup, a flavouring agent, and generally some drug as well, such as cascara or senna; the *syrupus aromaticus* (containing tincture of orange peel, cinnamon water, and syrup) is also termed *simple elixir*; the elixirs are official in the U.S. Pharmacopœia but not in the British; well-known examples are the *Elixir Cascara Sagrada*, the *Elixir Rhei*, and the *Elixir Sennæ*. *See* Prescribing.

Elster. *See* Balneology (*Germany, Saxony, Alkaline Waters*).

Elutriation.—The separation of the heavier from the lighter particles of a powder by suspending them in water, allowing them to settle, decanting off the supernatant fluid, allowing the remainder to settle again, and so on.

Elytro- or **Elythro-.**—In compound words *elytro-* (from *Gr.* ἔλυτρον, a sheath or vagina) means relating to the vagina; thus *elythritis* is inflammation of the vagina, *elytrocele* is vaginal hernia, *elytropexia* is vaginal fixation of the uterus, *elytroplasty* is vaginal plastic surgery, *elytroptosis* is prolapse of the vagina, *elytrorrhaphy* is narrowing of the vagina by a plastic operation, etc. *See also* Colpo-.

Emaciation. *See* Brain, Tumours of (*Symptoms, General Nutrition*); Intestines, Diseases of (*Malignant Disease, Symptoms*);

LEUCOCYTHÆMIA (*Symptoms*); LUNG, TUBERCULOSIS OF (*Constitutional Symptoms, Emaciation*); NEURASTHENIA (*Symptoms*); ŒSOPHAGUS (*Growths, Malignant*); PANCREAS, DISEASES (*Cancer, Symptoms*); RECTUM, DISEASES OF (*Cancer*); STOMACH AND DUODENUM, DISEASES OF (*Cancer of the Stomach, Symptomatology*); UTERUS, MALIGNANT TUMOURS OF THE.

Embalming.—The prevention of decay in dead bodies; nowadays it is carried out by injecting various fluids into them, such as solutions of sodium chloride, alum, arsenious acid, glycerine, methyl alcohol, or boiling water.

Embelia.—The berries of *Embelia ribes* and *Embelia robusta* are used in dozes of 1 to 4 dr., to provoke the expulsion of tapeworm; they are official in the Indian and Colonial Addendum to the British Pharmacopœia of 1898.

Embolism. *See also* AIR-EMBOLISM; ALCOHOLISM (*Acute, Diagnosis from Embolism*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Embolism*); HÆMORRHAGE; HEART, MYOCARDIUM AND ENDOCARDIUM (*The Embolic Process*); HEMIPLEGIA; INFARCTION; LUNGS, VASCULAR DISORDERS (*Pulmonary Embolism*); PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Varicose Veins, Thrombosis and Embolism*); PUERPERIUM, PATHOLOGY (*Sudden Death, Embolism*); SPINAL CORD, MEDICAL (*Morbid Anatomy, Vascular Lesions*).—By embolism is meant the carriage and impaction of a foreign body in some part of the vascular system, the impacted substance being called an *embolus*. Almost any organ in the body may be the site of its arrest. Embolism may also occur in the lymphatic vessels. The clinical features and points of practical importance in connection with embolism in the brain, heart, lungs, retina, kidneys, intestines, liver, and spleen will be referred to under their respective headings. The object of the present article is to give a short general summary of the subject.

Varieties of Emboli.—The most usual composition is a small part of a thrombus or a fragment from an endocarditic vegetation. In rarer cases it is composed of a spicule from an atheromatous plate, air, fat, cells, which may be parenchymatous, as liver or marrow cells, or tumour cells, bits of tissue, pigment granules, bacteria, and animal or vegetable parasites. Air emboli will be separately discussed under injuries of the neck, and fat emboli will be described under that heading.

Site.—Emboli are carried along by the general blood-stream until they become impacted in a vessel too narrow to permit of their further passage. This usually takes place in the general arterial system, the pulmonary arterial system, or in the branches of the portal vein. Occasionally it is met with in the systemic veins, and is then called a retrograde venous embolism. An example of this form is the arrest of an embolus in the pulmonary vein, resulting from the presence of vegetations on the mitral valve or a thrombus in the left auricle. Reference must also be made to a form of atypical embolism, of which several examples have been described which have been found to be due to a *patent foramen ovale*. This allows of the development of embolisms somewhere in the arterial system, the embolus being derived from the right side of the heart or venous system, and getting into the arterial system without passing through the pulmonary circulation. The fact that this can occur serves to explain certain cases where the size of an embolus has excluded the possibility of its having passed through the pulmonary capillaries.

Results.—The results vary according to whether the embolus be aseptic or septic. If it be aseptic, the pathological changes and clinical features are simpler in comparison with the results following one of a septic nature.

In the case of a *simple embolism* the blocking of the vessel is followed by necrosis of the tissue whose blood-supply is arrested. The necrosed area is known as an infarction, the size and shape of which vary with the particular organ and size of vessel affected. The colour is opaque, white, or yellowish, unless hæmorrhage is added to the necrosis, when it is described as hæmorrhagic. In the kidney and the retina the infarct is nearly always pale, while it is as constantly hæmorrhagic in the lungs and intestines. In the spleen and the heart it may be either red or white. When pale the periphery of the infarction is congested. The engorgement, whether peripheral or general, depends on a re-establishment of the collateral circulation and not on a venous reflux. The proper tissue elements of the organ undergo necrosis, the rapidity varying with the sensitiveness and vitality of the tissue. Later, the degenerated parts undergo absorption, with the formation of a cicatrix resulting from the inflammatory change which develops all round the infarct. In the case of infarction in the brain and spinal cord there is not a sufficient quantity of coagulable substance to make the area hard, and the parts undergo colliquative necrosis.

The results of a septic embolus are the formation of a small pyæmic abscess. In cases of acute infective endocarditis an *embolic aneurysm* is sometimes produced, most commonly in the cerebral or mesenteric arteries, this aneurysm being due to the action of the bacteria on the inner wall of the vessel.

Clinical Features.—These necessarily vary widely; embolism of the coronary arteries may produce almost instantaneous death, while on the other hand an embolism of the spleen may produce hardly any symptoms, and may have no clinical significance whatever.

Some cases have been recorded in the thoracic and abdominal aorta: in the former cases, sudden death usually resulted; in the latter,

paraplegia and other symptoms dependent on complete cutting off of the blood-supply from the lower extremities. The symptoms in cases of embolism of the extremities will vary according to the site of the affected vessel, and though rare, are readily recognised by the sudden onset and local disturbances induced. In some of these cases it is sometimes exceedingly difficult to distinguish between embolism and thrombosis.

Embolus or **Embolon.** *See also supra.* Derived from the Gr. ἔμβολος, a stopper or plug, the name *embolon* or *embolus* signifies the solid or semi-solid mass carried by the blood-stream into a vessel (usually an artery), which it obstructs. It may be part (or the whole) of a thrombus (*e.g.* a vegetation on a valve of the heart or on an atheromatous patch in the aorta), or fat, or air, or a mass of tumour cells, or a piece of syncytium from the placenta, or an animal or vegetable parasite, or a foreign body. The name *Retrograde Embolism* is given to the condition in which the embolus is carried in the opposite direction to that of the normal blood-flow (*e.g.* in the inferior vena cava in tricuspid incompetence). *Crossed or Paradoxical Embolism* is where emboli coming from the systemic veins pass through a patent foramen ovale and, having thus reached the left auricle and ventricle, are sent out by the systemic arteries.

Embrocation.—A liniment or other liquid medicinal application applied to the skin by rubbing or (less commonly) by painting, in order to remove or mitigate disease; it usually contains oil, camphor, or alcohol. *Roche's embrocation* is a patent medicine, said to contain tincture of ambergris, oil of cloves, and olive oil, used in whooping cough. *See* Linimenta; Prescribing; etc.

Embryectomy.—The removal of the embryo or fœtus and its annexa in cases of ectopic gestation; it is usually performed by abdominal section. *See* Ectopic Gestation.

Embryo.—The new organism in the uterus before the sixth week of pregnancy in the human subject, or, less accurately, the offspring of man or of an animal before its birth (Gr. ἔμβρυον, fruit of the womb, from ἐν, in, and βρύειν, to grow or swell); it is that part of the products of conception which is destined to form the new and independent being, and therefore, strictly speaking, it does not include the membranes or embryonic annexa. *See* Embryology; Fœtus and Ovum, Development; etc.

Embryocardia.—The name given to the condition of the heart sounds when there is dilatation and weakness (as in lobar pneumonia, typhoid fever, etc.); the long pause is greatly shortened and the first and second sounds become very similar (Osler).

Embryogenesis.—The development or formation of the embryo; in human embryology, embryogenesis occupies the first six weeks of antenatal life. *See* Embryology.

Embryography.—The study of development or embryogenesis by means of His' embryograph (a modified compound microscope for drawing large objects such as a complete embryo), or, simply, a synonym of embryology.

Embryology, Human.

See also Abortion; Fœtus and Ovum, Development of; Pregnancy, Pathology of (*Affections of the Ovum and Decidua*); Pregnancy, Pathology of (*Intra-Uterine Diseases*); Teratology. *See also* under the various organs, *e.g.* Generation, Female Organs of (*Arrested Developments*); Heart, Physiology of (*Embryology*); Palate (*Congenital Malformations of Mouth, Development*); Physiology, Reproduction (*Development*); Pregnancy, Multiple (*Twins*); Scrotum and Testicle (*Abnormalities*); Skin, Anatomy and Physiology (*Skin, Nails*); Teeth (*Development*); Uterus, Malformations of (*Development of Genito-Urinary Organs*); etc.

Embryology, if the word be used in a restricted sense, is the science which treats of the origin and formation of the parts and organs of the embryo. It is the study of the anatomy and physiology of embryonic life; for it has to do with the description of the parts which make up the embryo or its *anatomy*, as well as with the way in which these parts, often after passing through several intermediate forms, become the permanent organs of the embryo, or its *physiology*. It is not possible to separate the anatomical from the physiological part of the subject. It is quite possible, and indeed it is easy, to describe the organs and parts of the adult body, *e.g.* the individual bones of the skeleton, without referring to their functions, for they are fully formed and do not differ save in size at the various periods of life. To attempt, however, to describe the parts of the embryo is really to picture these parts as they appear at the various stages in the development of the embryo, in other words it is to represent their progressive elaboration. When an embryologist speaks of, say, the face of the human embryo, he is thinking of the five processes springing from the base of the primitive cerebral capsule, and gradually growing round the cavity of the stomodœum, and ulti-

mately uniting to form the facial framework; a living picture is as it were passing before his mind's eye. Embryology is, or ought to be, a kinematographic representation, a lifelike reproduction of the original moving scene of the whole embryonic period of antenatal existence. The embryo is not like a finished piece of machinery which can be studied both in action and at rest; it is unfinished, it is like a piece of machinery in process of construction, and its activities consist in a ceaseless progress towards a termination which shall be also a completion. In one sense (a limited one) the human embryo attains this finished stage with the close of the embryonic period of antenatal life; but in another sense (a wider one) it has not reached it even at the moment of birth, for some construction or organo-genesis goes on postnatally.

In a more usual and less restricted sense Embryology is the science which treats of all the vital phenomena which occur in the uterus and ovary before birth; in this sense, therefore, it includes fœtal physiology and germinal activity as well as ovulation and even menstruation. Its employment, however, in this extended sense is not to be commended. It is but one instance, although a very prominent one, of the general looseness of definition which has prevailed regarding the terminology of antenatal affairs; for instance, the name embryo has been applied to the new organism in the uterus both in the fœtal and in the embryonic period, while to the word fœtus the same wide significance has been given; again, to cite a still more evident example, the term ovum has been used not only for the female sexual cell both before and after impregnation, but also for the product of conception at the various months of intra-uterine life. Such a usage of terms is sadly lacking in scientific accuracy. In this article the word Embryology is employed as meaning the science which treats of the origin and formation of the parts and organs of the embryo;[1] it deals with a part of antenatal existence which, although somewhat short (five to seven weeks), is so crowded with events of such importance as to cause it to take rank above all the other periods of life, antenatal and postnatal. It includes the study of all the changes through which the embryo passes, from the appearance of its first rudiments in the embryonic area up to the transformation of the "transition organism" into the recognisably-human embryo or neofœtus.

General Principles of Embryology.

The general principles of Embryology lie buried somewhat deeply; they are far from the surface, and not easily to be reached or recognised when reached. It is difficult for the mind to picture a stage in the life of the organism when almost the only manifestation of vital activity is of the purely constructive kind, when, in other words, the framework of the body is being built up. So complex also is the process that certain portions of it, notwithstanding the laborious researches of keen and able embryologists, are still imperfectly understood or only guessed at. It is even more difficult to discover the laws which govern embryological processes, and to trace the general principles which underlie so complex a series of manifestations. One is almost driven to speculate whether, after all, there are laws regulating the mysterious metamorphoses of the embryo, whether indeed the parts do not fall together in kaleidoscopic fashion, and confusion happen somehow to give place to order. But reflection assures the investigator that there must be laws, otherwise he is forced to state that Embryology differs from all the other sciences in being uncontrolled by rule. Yet in no other part of biological study is it so difficult to trace and analyse the laws and their results. Events move so rapidly, form succeeds form and phase follows phase with such swiftness and with so much partial superposition, that nothing less than an arrestment of the succession of phenomena at various stages would serve to enable the embryologist to trace the procession of occurrences. Such an arrestment sometimes occurs, and pathology comes to our aid to help us to understand the intricacies of physiology; but the arrestment is usually of a local and incomplete kind, and its immediate effect is too often simply to increase the complexity of the problem we are attempting to solve. At the same time progress has been made, and further progress will yet be made by means of the careful study of these partial arrestments, which we may at once call teratological phenomena, and so ultimately the abnormal and the normal will prove mutually elucidative. In what remains of this section it is my purpose to try to educe from the embryological and teratological data at command such general principles and laws as may seem to be educible. Let the reader meanwhile bear in mind that some of these principles rest upon very insecure foundations, and that after all there is but little advantage in formulating statements which are apparently definite, concise, and clear-cut if the facts do not warrant them or if the facts are not sufficiently known to warrant them. Facts must not be twisted to suit general principles, but general principles must be allowed slowly and surely to emerge from the rigorous investigation and comparison of facts. The principles which follow may be said to be emerging from the crucible of modern exact research; they are not all pure gold, but they have appreciably been cleansed from some impurities.

[1] The word *Embryography* might perhaps be substituted, were it not undesirable to introduce new terms or terms used in new senses.

First, it may be stated with a large degree of confidence that the main progress of events and changes in Embryology is from the simple towards the complex and from the general towards the special; there is a trend towards elaboration, and from a homogeneity which is at any rate apparent even if it be not real there arises a heterogeneity which is both evident and real. From the comparatively simple-looking impregnated ovum comes the superlatively complicated embryo. From the successive divisions of that wonderful unicellular organism come all the tissues and organs of the still more wonderful multicellular organism. To the series of changes which are thus induced and in which we see the constant trend towards an increasing complexity the name *ontogenesis* has been given. It has, it is true, been employed in a somewhat wider sense ("the origin and development of the individual living being"), but it will be convenient to use it here as indicating the changes in the impregnated ovum by which it becomes a fully formed embryo. As this progressive differentiation is going on, generation after generation of cells comes forward and plays its part, being always represented by a yet more numerous progeny of cells, and so ontogenesis may be regarded as a great genealogical tree (Delage, *Les Théories sur l'hérédité*, 1895). In this genealogical tree, however, the successive generations do not coexist, but one gives place to the next and is indeed represented by it, for its cells have divided to form it.

Second, the heterogeneity which is produced in the process of ontogenesis is of two kinds. In one of these the differences are seen in the cells themselves; they grow unlike one another; there is a histological differentiation. In the other the differences are visible in the arrangement of the cells into organs; they are built together in different ways into organs; there is an organogenetic differentiation. There are different sorts of cells, and they are arranged in different sorts of ways.

Third, the startlingly divergent results found in the organs of the fully formed embryo may probably all be ascribed to differences in the rate of growth of the tissues composing the various organs and parts. In this way the manifold twistings and overlappings and separations and approximations which are seen in all embryos before their parts settle down into their permanent relationships are to be accounted for. A greater rapidity of growth of one part will cause a projection outwards or inwards at that part; a slowly growing part will find itself surrounded, built in, so to say, by the more active neighbouring areas; two organs at first far from each other may come into contact if both grow at a quicker rate than the body as a whole, they may even do so if one only grows faster, but then the meeting-place will not be the same. We speak in a loose fashion of the movement of various parts of the embryo in development, but the idea conveyed is erroneous if we mean by it that one organ without altering its rate of growth approaches another or passes away from it.

Fourth, there is a specificness in the ontogenesis of different types of animals. The embryos of different animals do not travel by the same route to reach their later stages; there is a wide diversity between their ontogenetic processes, how wide we do not probably fully realise as yet. Even when the fully formed organ or part in two species of embryo seems to be exactly similar, it does not apparently follow that it has reached this state by the same series of alterations. The embryo of the chick differs from that of the mammal in the details of its development; further, each has an amnion, but there is good reason to believe that it is not formed in the same manner in each. This principle cannot be too clearly kept in mind; for in the past it has been neglected, and what was observed in the chick embryo was regarded as proven also for the human embryo, with results which were disastrous so far as accuracy was concerned.

Fifth, as a general rule, the higher an animal is in the zoological series the more complex is its ontogenesis. At the same time the progress towards complexity need neither be uniform nor constant. The highest of one group of animals may have an ontogenesis much more complex than the lowest of the group next in order above it. There is a specificness even in this matter, and there is no complete chain of types, representing a regular sequence of ascents.

Sixth, there is a general principle which may be stated in several different ways, and which has given rise to an immense amount of discussion. It has also been called the "great law of biogenesis." It may be enunciated in this way — that ontogenesis never follows a simple and direct route, but reaches its end by detours ("ces détours de l'évolution," *Delage*). In the forming of the completed embryo there are stages passed through which apparently serve no useful purpose; there are organs which appear and disappear again leaving sometimes rudiments which confessedly are dangers to the organism; some of these transitory organs have a temporary use, but others seem to have none at all. The higher vertebrate embryo, for instance, has three sets of urinary organs—the pronephros and its duct, the mesonephros and its duct, and the metanephros and its duct; and yet the necessity for some of them is not apparent. Further, it has for long been noted that many of these "detours" or temporary organs and parts distinctly recall the stages or appearances seen in animals lower down in the zoological scale; and so it came to be thought that in ontogenesis there is a sort of recapitula-

tion of Comparative Anatomy, or, to put it in more modern terms, that ontogeny gives a condensed phylogeny, or that there is a reminiscent projection of phylogeny into ontogeny. Before, however, we discuss the correctness of this conclusion, let us see that we have a clear idea of what is intended by this general principle, this "great biogenetic law."

Henry Drummond[1] gave a picturesque and vivid description of what he understood and read into it. He wrote: "The human embryo is a subtle phantasmagoria, a living theatre in which a weird transformation scene is being enacted, and in which countless strange and uncouth characters take part. Some of these characters are well known to Science, some are strangers. As the embryo unfolds, one by one these animal actors come upon the stage, file past in phantom-like procession, throw off their drapery, and dissolve away into something else. Yet as they vanish, each leaves behind a vital portion of itself, some original and characteristic memorial, something itself has made or won, that perhaps it alone could make or win—a bone, a muscle, a ganglion, or a tooth—to be the inheritance of the race." So Drummond pictured the human embryo "wandering among the ghosts of departed types"; in making a man, "Nature introduced the framework of these earlier types, displaying each crude pattern by itself before incorporating it in the finished work." "Every creature that lives climbs up its own genealogical tree before it reaches its mature condition."

This law, which as we shall see is not so really a law as has been thought, may be stated in another way. Apparently the framework of the body is not laid down once for all, but is rather constructed in portions, and even in overlapping portions, so to speak. One part of the framework may be replaced in whole or in part by a new substructure often of a type quite different from that which has preceded it. Readjustment, adaptation, alteration, and variation are processes constantly at work in the life of the embryo; it is by reorganisation that order is brought out of the seeming chaos of the results of blastodermic activity. If one were to imagine a builder constructing a palace by first erecting the scaffolding for a cottage; by then, after building operations had only just been commenced, replacing this by the scaffolding for a villa; and that again by the framework necessary for a mansion; and then, finally rearranging, and as far as possible utilising, all the materials for the formation of the palace; one would have an idea, albeit an imperfect one, of the character and complexity of ontogenesis. The comparison is, of course, almost ludicrously inadequate, for it gives no indication of the marvellous manner in which in organogenesis all the materials of the temporary scaffoldings are made use of in the construction of the permanent edifice, are worked into it so exactly as to be indistinguishable from each other in it. Only here and there in the body, in the appendix vermiformis, the fossa ovalis, and the ductus arteriosus, are to be seen the traces of some parts of the earlier formations which have either been incompletely utilised or have been imperfectly replaced by the later productions.

This same general principle of ontogenesis may be set forth in yet another way, as evolution by atrophy. In order that a new scaffolding may be set up it is necessary that the former atrophy; if a new plan of construction is to be followed it is needful that the old plan be abandoned. To quote from Ernest Mehnert[2] (as rendered by J. Arthur Thomson[3]), involution and evolution go hand in hand. The ovum develops, the polar bodies degenerate; the blastoderm develops, the merocytes degenerate; the head grows and the tail dwindles; the higher nerve centres of the mammalian brain increase and the occipital region diminishes; the metanephros comes and the pronephros goes; the backbone strengthens and the notochord disappears; and so on throughout a continual necrobiosis.

In whatever way it may be stated, there can be no doubt that in ontogenesis there is very evidently this principle of reaching the final result by ways which appear to be roundabout and by scaffoldings that are temporary. When, however, the attempt is made to find a constant and determining rule or law dominating the ontogenetic detours and the developmental scaffoldings, difficulties at once emerge; the explanation that the embryo of the higher vertebrates passes rapidly through a series of stages which represent the permanent forms of embryos lower in the zoological scale breaks down at once upon close examination, and as yet no other satisfactory hypothesis has arisen to take its place. But this fact demands a separate paragraph, and it will be best to embody it in a general principle, the *seventh*.

Seventh, then, it may be affirmed that ontogeny does not give a short recapitulation of evolutionary progress; it is not an epitomised phylogeny. This is seen in the fact that the time at which an organ appears in individual development does not always indicate the historical or phylogenetic age of that structure. If there is, then, a recapitulation in ontogenesis it is one in which Nature makes mistakes; or, to express the matter in more scientific language, there are "time-displacements" in development. For instance, the heart is regarded as a secondary differentiation (due to functional adaptation) of

[1] Drummond, H., *The Ascent of Man*, p. 87, 1894.

[2] Mehnert, E., *Biomechanik erschlossen aus dem Principe der Organogenese*, Jena, 1898.

[3] Thomson, J. A., "Mehnert's Principles of Development," *Natural Science*, xiv. 385, 1899.

that part of the primary ventral blood-vessel where the greatest resistance has to be overcome; but in the organogenesis of the human subject the heart appears before the associated main vessels. The mammary glands, also, make their appearance in the individual before the sebaceous glands from which they are supposed to have been differentiated; and the paired eyes in vertebrates arise long before the much older pineal eye. It would seem, to revert for a moment to a non-scientific way of speaking, as if Nature in telling in the individual the story of his descent (or ascent) made occasional slips and put incidents in a wrong order of occurrence. Doubtless the mistakes are only seeming mistakes, and an explanation is hidden away somewhere if it could but be found. Possibly, as Mehnert suggests, when an organ rises in physiological importance and structural differentiation it acquires a proportionate increase in its rate of development. Progressive organs are accelerated in their rate of development, and retrogressive ones tend to be belated; and their state of acceleration or belatedness (or retardation) is due to the intensity of developmental energy in them. It is not yet clear what exactly it is that exalts or depresses developmental energy. It must apparently be conceded, therefore, that ontogeny is not in point of the sequence of events a recapitulation of phylogeny; but it does not necessarily follow that there is no tendency for it to be so. It would seem that there is a constant inclination towards a chronological parallelism between ontogeny and phylogeny, along with frequently occurring interruptions and disturbances due to circumstances possibly of an environmental or hereditary character. It is not true then that "every creature climbs up its own genealogical tree," but it would seem to be true that it tries to do so; perhaps it sometimes swings from branch to branch! At the same time, even if the recapitulation principle be accepted in this partial form, it must not be forgotten into how marvellously short a story has been condensed so inconceivably long a history. In the first six weeks of the antenatal life of the human embryo are represented in epitome all the ages that have gone to the making of man! And each life adds on something to the ever-lengthening tale!

Further than these seven principles it does not seem wise in the present state of our knowledge to go. It would be easy, extremely easy, to add to the number, but the additions would be hypothetical generalisations, and they would also be hypotheses about which embryologists are far from being in agreement. There is, for instance, the question whether the various parts of the embryo exist already in the ovum and need only to be rearranged during organogenesis, whether each organ, each tissue, and each cell is represented by a distinct rudiment (*anisotropy* of the ovum); or whether the substance of the ovum is homogeneous and contains no parts specially set aside to form definite structures in the embryo, whether it is *isotropic*. The weight of evidence is apparently in favour of the isotropic character of the ovum; but it would seem as if in certain animals it were sometimes, and in others always, anisotropic. The evidence is chiefly of an experimental kind, and is not free from fallacy. At any rate, it is obvious that under the circumstances it would be unwise to attempt to formulate a general principle on the data at command. The same remark applies to the problem of the causes and manner of the differentiation of parts in the embryo, to that of morphogenesis, to that of regeneration, and to many other biological questions.

Enough has been said to show that there are general principles in Embryology, general laws, the appreciation of which will facilitate the understanding of the architectonic phase of antenatal life. The outstanding peculiarity of Embryology is still its complexity, for the statement of general principles does not lessen that—it rather throws it into bolder relief; but surely behind this complexity there is a real simplicity if we could find out the great determining factor in embryo-formation. The results of the action of gravity are complex enough, but behind their perplexing manifestations is the simple law of gravitation. There may be some such simple and universally applicable principle of ontogenesis, if it could only be discovered. We need for Embryology an Isaac Newton "of an extraordinary genius and proficiency in these things."

Chronology of Embryonic Life

In this section and in those that follow it an attempt is made to give a chronological sketch of the phenomena of embryonic life. The period of antenatal existence from the appearance of the first rudiments of the embryo in the embryonic area up to the transformation of that embryo into the neofœtus is dealt with. The neofœtal period is the transition time intervening between embryonic and fœtal life. Its leading character—gradual assumption by the embryo of a recognisably human appearance—will be described later.

It is with the embryo, then, before the neo fœtal epoch that we have to do in these sections. The terminus of embryonic life we fix at the end of the sixth week of intra-uterine existence. But at what time may it be said that the typically embryonic life of the new organism begins? We have announced the *terminus ad quem*, but what declaration have we to make regarding the *terminus a quo*?

It may be admitted at once that it is not yet possible to fix exactly the date when human embryonic life begins and germinal existence ceases. If we regard the antenatal life of forty

weeks as beginning with the impregnation of the ovum by the spermatozoon, then true embryonic life must manifestly begin at a date later than that. How much later, however, we are not in a position to state. If we allow a week, we are probably giving too much; if we decide upon less than a week, we may not be giving enough. In the absence of exact information, shall we say a week? Shall we let it be understood that provisionally we regard the beginning of truly embryonic life as coinciding with the beginning of the second week of antenatal life, reckoning from the date of impregnation of the ovum? I am reminded that we do not know at what time subsequent to coitus impregnation occurs. That I know well, and I have, therefore, in my scheme of the divisions of antenatal life (Fig. 1), set apart a period of half a week during which the delegated spermatozoon is presumably travelling towards the destined ovum. I make the supposition that three and a half days after coitus impregnation occurs and antenatal life in its unified form (*i.e.* within the walls of one cell) begins; a week later I presume that embryonic existence commences with the appearance of the first rudiments of the embryo in the embryonic area; and in five weeks after that I assume that typically embryonic life is over and neofœtal existence has begun. This conception is shown in schematic form in Fig 2.

These exact periods of time are not, of course, insisted upon. They are advanced only as possibilities, which may turn out to be probabilities, but which may also prove to be quite otherwise. I do not indeed think it likely that it will be found that the whole period of antenatal life in the ordinary sense of the term (say, forty weeks or two hundred and eighty days) is an exact multiple of embryonic existence. Our knowledge of Nature's operations and mode of working out her results does not lead us to expect that she deals with exact multiples, as we understand them. Nature, we can well believe, in her calculations is not limited to the simpler exercises of arithmetic; she can deal with fractions!

Let us commence our study of the chronology of embryonic life by realising how important it is that we should form in our mind a visual image of the human embryo in the various weeks of its kaleidoscopic existence. It is a most difficult task; nevertheless, however difficult it is, it must be attempted, else all efforts to form rational ideas regarding the pathological phenomena of these embryonic times must end resultless or in error. In the later weeks (*e.g.* the fifth and sixth) of embryonic life, the difficulties are not so overpowering, for observations of human embryos of these ages are not wanting, and conclusions may fairly be drawn from them. But in the early weeks! We know almost nothing of human blastoderms of less than seven days' age, we know but little of embryos of the second week, and even up to the end of the first month embryological facts are scanty. The *quæsita*, indeed, are many, but the *data* are few. So the embryologist has been driven to study the early stages in the lower animals, and has not always been careful to distinguish between the occurrences which are met with in them, and those which there is good evidence to show take place in the human embryo. In the text-books the details of Comparative Embryology are often interpolated among those of Human Embryology; and writers, in transcribing from original sources of information, are oftentimes careless about distinguishing between what has been found, for instance, in the embryo chick, rabbit, or pig, and what in the human subject. All this is very discouraging, and, in order to correct many misapprehensions, it would be well worth some embryologist's time and trouble to edit a text-book in which all the facts known certainly regarding the *human* embryo were printed in red letters. I do not depreciate the value of Comparative Embryology, but I deny that its data can be unquestioningly transferred to fill up gaps in our knowledge of Human Embryology. Human Embryology so constructed is simply a series of scanty facts laced together by a series of assumptions.

The Blastocyst in the Second Week after Impregnation

A. *At the Beginning of the Second Week*

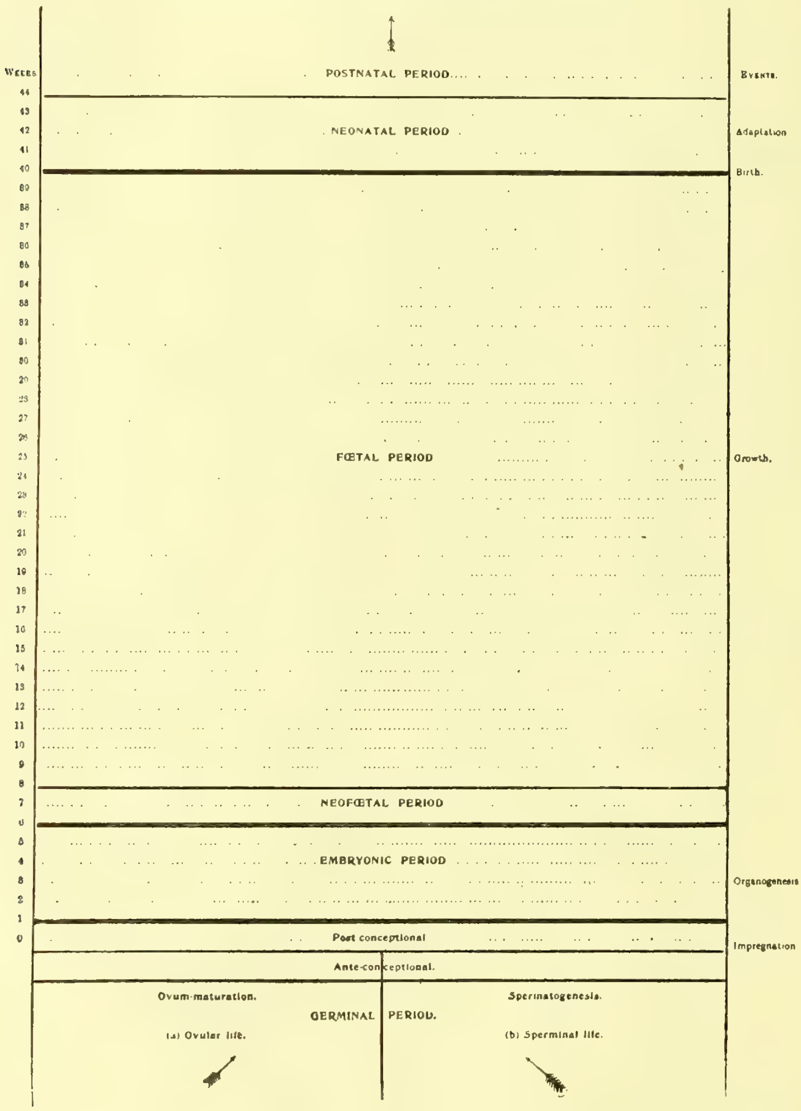
What mental picture can we form of the human blastocyst as it exists during the second week after impregnation? Two things, at least, we may rest well assured of: (1) the appearances of the blastocyst at the beginning and end of this week will be very different, and (2) the embryo will, in size and apparent importance, seem almost insignificant in comparison with its annexa and the decidual membranes. Development is very rapid in these early days, and a week is a long time in respect of the changes which are effected in it. At a later date than the second week, the truly embryonic part of the new organism is little more than an appendage of its own vesicles and membranes, and it may be concluded that this character is still more emphasised at the time with which we have here to do. Of these two things we feel sure within measure, but of what else?

Let us try to picture to ourselves what has taken place towards the close of the first week after impregnation. The impregnated ovum, now doubtless a blastodermic vesicle or blastocyst, has found its way into the uterine cavity. In so doing it has not dropped into an open space, as diagrams deceitfully suggest to the mind's eye, but has insinuated itself between uterine walls in complete contact, and possibly may have had some difficulty in thus making its way. It passes some distance from the uterine orifice of the Fallopian tube down which it has

come, perhaps creeping on amœba-like by means of the plasmodial syncytium with which it is covered, perhaps waved onwards by ciliary action aided by a partial uterine contraction, perhaps lifted forward by a sort of congestive elevation mucosa under the margin of which it can edge itself and so get below the surface; perhaps it simply rolls into the furrow between two ridges and lies there till the swelling ridges meet over it and shut it in; perhaps it falls astride a

FIG. 1.

THE DIVISIONS OF ANTENATAL LIFE.



of the mucous membrane upon which it is resting (who can say?); at length it reaches its destined anchorage. But what is it that constitutes a suitable mooring place, and by what means is the anchorage rendered tenable? Perhaps there is found an area of mucous membrane without its epithelial covering (or its cilia) where the blastodermic vesicle can reach connective tissue; perhaps there is a little slit in the ridge, and projecting downwards on each side of it fills also the adjacent furrows, and so remains till neighbouring parts grow up and around it; perhaps it bores its way downward through the epithelium by the destructive or phagocytic action of its covering cells, and the little opening through which it passes is closed with a coagulum. It is easy to multiply suppositions. Curiously enough one of the most obvious sug-

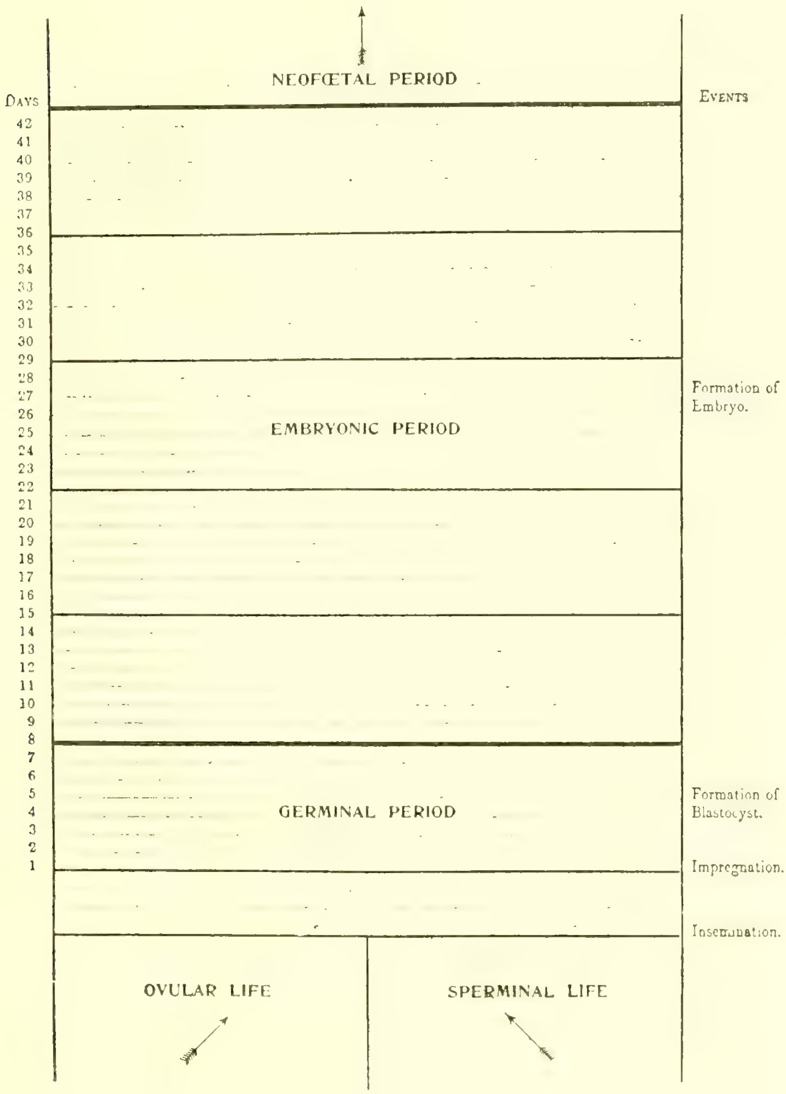
gestions—that the ovum passes or sends processes into the mouths of the uterine glands—is that which nearly all embryologists nowadays agree in rejecting, and that apparently on good grounds.

therefore, still use the term decidua reflexa, without, of course, holding the Hunterian view of its formation.

H. Peters' specimen (*Ueber die Einbettung des menschlichen Eies*, Leipzig u. Wien, 1899) gives

FIG. 2.

THE DIVISIONS OF EARLY ANTENATAL LIFE.



According to the view we take of the manner of mooring of the ovum in utero will be the theory we hold of the mode of formation of the decidua reflexa (*s. capsularis*). John Hunter's conception of its origin has been abandoned, but there seems no reason sufficient to warrant the abandonment of the name which he gave to it; names in medicine do not always retain their original and etymological meaning. I shall,

us the means of imagining how the reflexa is formed, for its age was five or six days, and this decidual membrane was still in process of differentiation. The specimen showed the ovum entirely sunk in the compact layer of the decidua vera about the middle of the posterior wall of the uterus. Over each side of the ovum was an extension of the vera forming the outermost portion of the reflexa, and the central portion

was made up of a mass of fibrin. It may be supposed, therefore, that the ovum, after sinking down into the decidua vera, begins to excavate laterally as well as deeply; and so it comes about that the overhanging rim of the mucosa (the vera) forms the reflexa, while the opening made by the ovum is plugged with a coagulum of fibrin. In Leopold's earliest specimen (*Uterus und Kind*, Leipzig, 1897), the age of which may be stated as the end of the first week, the reflexa is seen surrounding the ovum entirely, but it is some of which communicate with lacunæ in the tissue surrounding the ovum (the trophoblast). In the spongy layer are small arteries. Large polymorphic cells are found in the compact portion, possibly decidual and maternal in nature, possibly also trophoblastic and fœtal. There is a loosening of epithelium going on in the glands of the serotina. In Leopold's specimen the serotina immediately below the ovum has half the thickness of the mucous membrane in its near neighbourhood; the surface capil-

FIG. 3.—Section through Peters' early ovum, lying imbedded in the uterine mucosa. *K.A.*, the embryo with amniotic sac and umbilical vesicle; *U.E.*, uterine epithelium; *Cap.*, decidua reflexa (capsularis); *Tr.*, trophoblast; *Ca.*, maternal capillaries; *Dr.*, glands; *Bl.L.*, blood-pools; *Comp.*, compact layer of decidua; *M.*, mesoderm; *G.P.*, spongy tissue. (After Peters.)

thin and fibrinous in its polar portion, and the outer surface of the ovum is intimately attached to it (Fig. 5). The differentiation of the mucosa forming the decidua vera into a compact (surface) and a spongy (deeper) layer is quite evident at this age; in the reflexa near its base are some glands lined with cubical epithelium, in the interglandular tissue are some large deeply-stained cells (maternal or fœtal?), and there is no epithelium on the inner aspect of this reflexa.

The mucous membrane to which the ovum is moored or anchored is the serotina, now often called the decidua basalis or placental decidua. It consists, even at the age of Peters' specimen, of a superficial compact layer and a deeper spongy one. In the former part are signs of œdema with dilated capillaries and blood spaces, laries open into the spaces (intervillous) between the processes of the trophoblast, so that even at this early date maternal blood bathes the outer aspect of the ovum.

In this manner, therefore, or in a manner somewhat like it (for it is unwise to draw deductions hastily from the interpretation of so few specimens), the fertilised human ovum becomes anchored to the uterine wall. The term *anchorage*, however, hardly meets the descriptive requirements of the case, when we think of the ovum surrounded as it is on all sides by maternal decidual tissues; it will be more correct to speak of it as planted in or imbedded in the uterine mucous membrane.

Let us now construct our mental picture of the ovum itself, as it lies thus imbedded at the

end of the first week after impregnation. At this date it consists evidently of two very unequal parts, one of which may be called extra-embryonic and the other embryonic. Let us look first at the former part, which is not only much the larger of the two, but is also apparently of vastly greater importance. The ovular mass (blastodermic vesicle, blastocyst, chorionic vesicle, or whatever other name is given to it) has measurements varying from 1·6 : 0·8 : 0·9 mm. (Peters' specimen) to 4·0 : 3·7 mm. (Leopold's specimen); it is, therefore, a lenticular mass which lies thus in the uterine mucosa. Its outer aspect consists of a thick layer of what may be conveniently termed trophoblast, which has been probably developed from the epiblast of the ovum. In this trophoblast layer are numerous lacunæ and vacuoles, some of which, according to Peters, contain maternal blood. The processes or strands between the lacunæ consist of nucleated cells with distinct outlines, and near their outer ends some of them divide irregularly. In some parts the blood lacunæ are lined by a nucleated protoplasm in which no cell-outlines are visible, syncytial, therefore, in nature (Peters). Much difference of opinion exists as to the mode of formation of the syncytium which plays so important a part in the union between maternal and ovular tissues. Perhaps the most likely view is that it is formed by the action of the maternal blood upon the trophoblast cells; much of the protoplasm of the syncytium may then be of maternal origin, while its nuclei are the changed nuclei of the trophoblast (*vide* Bandler's article, *Amer. Journ. Obst.*, xlvi. 145, 1902). The trophoblast, it is thought, invades the compact layer of the uterine mucosa, while the maternal blood has a corrosive action upon the trophoblast cells, converting them into syncytium. It is to be noted that as yet there are no villi, but here and there processes of trophoblast make their way into blood sinuses in the decidua. We may provisionally call the interspaces intervillous. Beneath the trophoblast is a thin layer of mesoblast, but the latter scarcely passes at all into the processes formed by the former. In Leopold's early ovum there is a later stage of the development of the union between ovum and decidual membranes. In it the ovular mass is covered with villi (save in one small part); some of these are branched, some simple; some have a mesoblastic tissue core, others are purely buds of syncytium; some merely reach the decidua (serotina and reflexa), others are slightly imbedded in it; some show capillary formation in progress in them, others do not. Probably the villi in Leopold's ovum are the transformed strands of trophoblast seen in Peters' specimen. Between the villi are to be seen heaps of decidual cells, the beginnings, no doubt, of the trabeculæ which are found in later stages of development. In the intervillous spaces, also, maternal blood circulates freely, for maternal capillaries open into them.

It is to be noted then, that even at so early a period as the end of the first week after impregnation, the ovular mass is surrounded by a sort of primary placenta. The epiblastic proliferation or trophoblast would seem (as J. C. Webster states in his *Human Placentation*, p. 63, 1901) to have the following functions: it acts, first, as a means of attaching the ovum to the mucosa; second, its strands probably serve as pathfinders for the future permanent villi; third, it probably absorbs nourishment for the ovum from the decidual tissues and from the serum and blood with which it comes in contact. It is possible also that fragments of its syncytial covering may be broken off and travel into the maternal circulation (Bandler, *New York Med. Journ.*, lxxvii. 54, 1903), a possibility which takes on great importance when considered in connection with the development of deciduoma malignum and the pathology of pregnancy (*e.g.* the causation of *eclampsia*, *q.v.*). The trophoblast, it may be added, may be regarded as the same structure to which the names of chorion, exochorion, primitive chorion + decidual layer, epiblast of non-embryonic part of blastodermic vesicle, and ectoplacental formation have been given by various writers. It is matter for great regret that there is such a plurality of names; it is probable that the term trophoblast will gradually supersede the others, with benefit to the

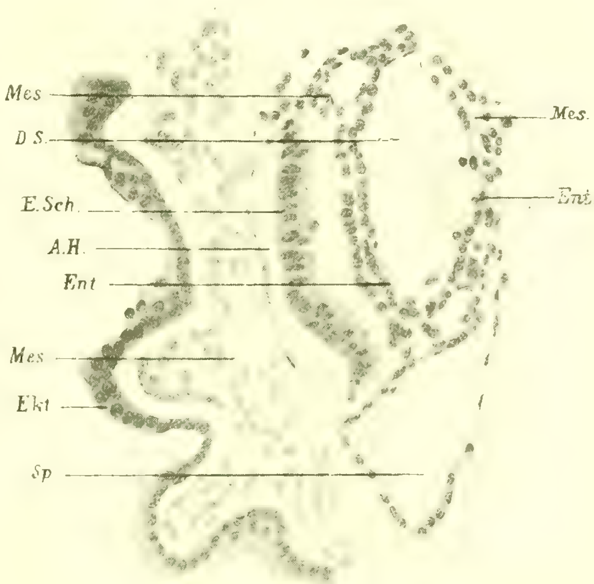


FIG. 4.—Section through embryonic part of Peters' early ovum. *A.H.*, amniotic sac; *D.S.*, umbilical vesicle; *E.Sch.*, embryo; *Ekt.*, ectoderm; *Mes.*, mesoderm; *Ent.*, entoderm; *Sp.*, fissure in exo-cœlom (?). (After Peters.)

reader who does not wish to solve troublesome terminological puzzles.

Let us now proceed with our inquiry regarding the constitution of the ovular mass at the end of the first week after impregnation. What lies inside the trophoblast? As has been said, there is inside it a thin layer of mesoblast (two to four thicknesses of cells, *Peters*), a little thicker opposite the pro-embryon. Its cells are rounded, oval, and spindle-shaped. Inside the sac formed in this way by trophoblast lined with mesoblast is situated at one point the pro-embryon (for we can scarcely yet call it "embryo"). Here between the mesoblast lining

Fig. 5.—Section through Leopold's early ovum (end of first week of pregnancy). *M.*, muscular wall with ends of glands (*de*) in it; *D.v.*, decidua vera, with compact (*Comp.*) and spongy (*Amp.*) layers; *l.c.*, leucocytes in the lymphatics; *D.c.*, decidua capsularis or reflexa, in which up to *dr. cp.* gland spaces are seen, and above *i i* it consists mainly of fibrin; *b b* is the ovum or chorionic sac resting on a spur of the decidua; *a*, amnion; *d*, decidua serotina; *e*, mouth of maternal vessel; *h*, attached villi; *f*, imbedded villi. (*Vide*, Leopold's Atlas, *Uterus und Kind.*)

the trophoblast and the epiblast of the pro-embryon is placed a small cavity, that of the amnion. It is completely closed, and is lined with a layer of very flat cells opposite the pro-embryon and with the cylindrical cells of the embryonic region themselves. It is to be noted that in this earliest known specimen *(Peters')* the amniotic sac is already closed, so that we can know nothing of its mode of formation; but the fact of its being closed suggests the question whether it was ever open. Probably the amnion in the human subject is not formed by the up-heaval of folds of extra-embryonic somatopleure at all, but by a breaking down of epiblast tissue to form a cavity (Berry Hart), or by inversion of the blastoderm (Mall [1]). At any rate we have to put an amniotic cavity into our mental picture of the ovular mass at the end of the first week after impregnation. On the side of the amniotic sac next to the centre of the ovular mass is the epiblast of the pro-embryon, and to the inner side of it again is the umbilical vesicle lined with hypoblast cells and showing also some mesoblast cells outside these. In the pro-embryon or embryonic area, it is to be noted there is as yet no appearance of the primitive streak, so that an amniotic sac and an umbilical vesicle are in existence before any trace of the embryo itself appears. This, also, seems to be the state of affairs in F. P. Mall's early ovum (*Johns Hopkins Hosp. Bull.*, iv. 119, 1893). Neither is there any indication of a cord-like structure or Bauchstiel, for as yet the amniotic and umbilical sacs are, as it were, buried in the mesoblast of the chorionic vesicle. Graf Spee, who carefully examined Peters' specimen, could not be sure which was the cranial and which the caudal end of the pro-embryon. The remaining space inside the ovular mass is the extra-embryonic cœlom or simply the exocœlom.

Such then is the conception we have to form in our mind of the new organism in the uterus about the end of the first or the beginning of the second week after impregnation. It is, so to say, only a peep that we thus obtain of antenatal affairs at this early period. There is a great lacuna in our knowledge, a lacuna which extends from the time when the human ovum leaves the ruptured Graafian vesicle up to the time when it is found implanted in the uterine mucosa with its amniotic sac and umbilical vesicle closed and the decidua reflexa nearly completed. This gap in our knowledge we may attempt to fill up by observations upon the lower animals, but it is not safe to conclude that what occurs in them will occur in the human subject. We may also attempt to supply the missing parts of the kinematographic representation of the progress of formative processes from our imagination, but it is still less safe to pursue this plan. So we must be content to wait till yet earlier specimens of human ova are met with. It is the safest method, but the waiting may be long. Better, however, is it to wait for facts than to construct theories which have soon to be demolished.

[1] Mall, F. P., "Development of the Human Cœlom," *Journ. of Morphol.*, xii. 395, 1897.

B. *At the End of the Second Week.*

In order to picture to ourselves the state of affairs in utero towards the end of the second week after impregnation, we have the specimens of Graf Spee (12 days), Merttens (8 days (?) but probably more), F. Burgio (12 to 13 days), Reichert (12 to 13 days), Breus (12 to 14 days), F. P. Mall (13 days), Keibel (13 to 14 days?), Merttens (14 days), His (13 to 15 days), Leopold (14 to 15 days), Eternod, and some others.[1] Of course it must be remembered that these ages are only approximate, for it is extremely difficult from the history of the case to fix the probable age of the conception. Further, the appearances and measurements of the embryos cannot be used as definite guides, for there is some reason to believe that all embryos do not progress towards a given stage in their development with the same degree of rapidity. With these considerations in full view we may now proceed to form an idea of the condition of the new organism at the end of the second week.

At this date there exists, as all the specimens show, a distinct and recognisable Embryo; no longer have we to do with a pro-embryon or embryonic indication. At the same time, the embryonic part of the ovular mass is still insignificant in point of size when compared with the embryonic vesicles and the decidual membranes. We shall, therefore, first consider the decidual membranes, the chorion, the amnion, and the umbilical vesicle.

The decidua reflexa (capsularis) now forms a complete fold over the ovular mass, but the outer polar portion is mainly, if not entirely, fibrinous in nature, showing neither surface epithelium nor decidual cells, nor glands. The decidua serotina (basalis) exhibits clearly the division into a compact and a spongy layer. The outer parts of some of the glands are obliterated. Large decidual cells (many of them spindle-shaped) are seen in the compact layer, as are also many sinuses containing blood. Nucleated plasmodial masses are to be discovered lying among the decidual cells. In the spongy layer the gland spaces are large, and are lined with columnar or cubical epithelium. Decidual cells and plasmodium are also found in the spongy part. On the surface of the compact layer of the serotina next to the ovular mass is plasmodium or syncytium continuous with that covering the villi (for there are now distinct villi). The general opinion held at the present time is that this plasmodium is not formed from the orginal surface epithelium of the decidua or from that of the gland ducts, but in the way which has been already indicated.

Inside the decidual membranes (reflexa and serotina) lies the ovular mass or chorionic vesicle (as we may now call it). It has a diameter varying from 3 to 5 mms., and it is furnished at this date with recognisable villi, no longer with simple strands of trophoblast. These villi are mostly simple, but a few are branched, and they may cover the whole chorionic vesicle or be defective at the poles; they measure about ·5 mm. in length; and they are attached to the decidua reflexa and serotina. They are covered with a coating of syncytium, and below that lies a layer of distinct cubical cells, Langhans' layer. Nearly all the villi have now a mesoblastic core of connective tissue. The syncytial covering of the villi is, as has been already stated, continuous with that on the surface of the

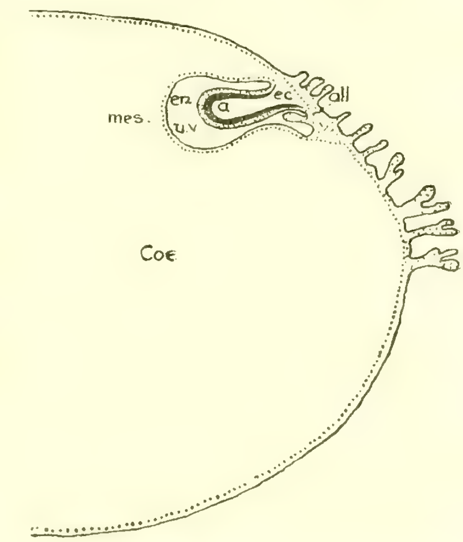


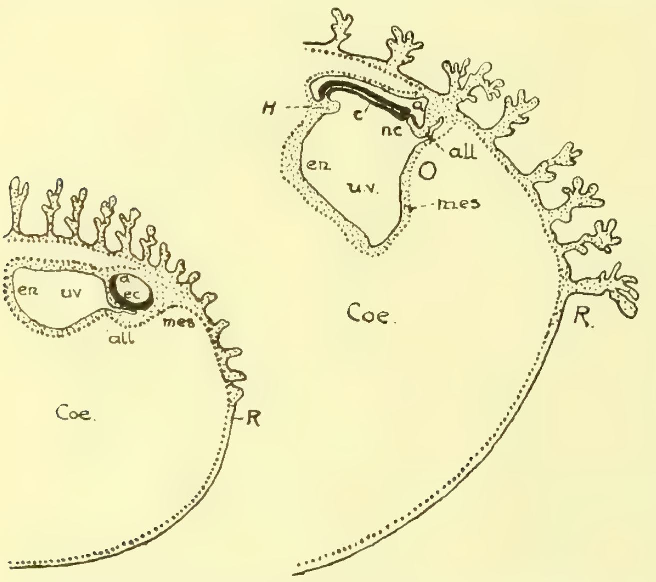
FIG. 6.—Diagrammatic section of one-half of Mall's early ovum. Enlarged 10 times. The villi are only shown in part. *Ec.*, ectoderm; *en.*, entoderm; *mes.*, mesoderm; *u.v.*, umbilical vesicle; *coe.*, cœlom; *all.*, allantois; *a.*, amnion. (After Mall.)

serotina and reflexa. There are distinct intervillous spaces containing maternal blood, and into these spaces masses of syncytium here and there project. About this date (end of second week) capillaries are found in the mesoblastic core of the villi. At the point of attachment of a villus to the decidua there is usually a great proliferation of the cells of Langhans' layer, forming a distinct knob.

What now are the contents of the chorionic vesicle at this age? Attached to the inner aspect of the chorionic vesicle by a stalk (Haftstiel, Bauchstiel, anlage of the umbilical cord) is a mass which consists of the amniotic sac, the umbilical vesicle, and the embryo (Figs. 6, 7, and 8). The amniotic sac is closed, and it covers the dorsal surface of the embryo lying close to it; on the ventral surface is a wide opening leading from the hypoblastic or entodermal interior into the umbilical vesicle. Both the amniotic sac and the umbilical vesicle are

[1] The references of the contributions in which these embryos are described will be found in Hertwig's *Handbuch der vergleichenden und experimentellen Entwickelungslehre der Wirbelthiere*, Jena, 1901-1903, and in other recent text-books of Embryology, *e.g.* that of M'Murrich.

small structures in comparison with the chorionic vesicle. The embryo, measuring about 1·5 mm. in length, consists of a mass of cells lying between the amniotic sac and the umbilical vesicle, and continuous at its caudal end with the Haftstiel or umbilical stalk. In it can be recognised a primitive streak (thickening of epiblast cells), and in front of it a canal (the neurenteric) by which the amniotic sac communicates with the archenteron which is now showing signs of division into an intra-embryonic part (the gut tract), and an extra-embryonic part (the umbilical vesicle). In front of the opening of the neurenteric canal is the medullary plate with the two medullary folds forming the medullary groove which is not yet converted into a canal. These parts are formed by a thickening and an infolding of the epiblast; similarly on the ventral surface there is an infolding of hypoblast which gives rise to the notochord, which is at first a hollow rod of cells communicating at its posterior end with the neurenteric canal. In some of the specimens said to be of the age we are now considering, namely, end of second week after impregnation, there are indications of five or seven protovertebræ (the primitive segments of Minot, *Urwirbel* of the Germans) in the mesoblast near the notochord. There may also be a trace of the anlage of the heart near the cephalic end of the embryo in the form of two tubes in the mesoblast. According to Graf Spee there is, about this time, a small diverticulum from the single cavity of the gut tract and umbilical vesicle into the Bauchstiel, which he regards as the beginning of the allantoic duct (Fig. 8).



FIGS. 7, 8.—Diagrammatic section of two early human ova. Enlarged 10 times. About half of each is represented, and the villi are outlined over only a part of the ovum. *R.*, Rauber's layer; *a.*, amniotic sac; *u.v.*, umbilical vesicle; *ec.*, ectoderm; *en.*, entoderm; *mes.*, mesoderm; *all.*, allantois; *c.*, notochord; *n.c.*, neurenteric canal; *H.*, position of heart. (After Graf Spee and Mall.)

Vessels also can be recognised in the wall of the umbilical vesicle; it is not clear that these yet communicate in any way with the heart rudiment to constitute a vitelline circulation. In Eternod's specimen (*Anat. Anz.*, xv. 181, 1898) there was a curious, hitherto undescribed vein in the caudal part of the umbilical vesicle ("anse veineuse vitelline"); there was also a single allantoic vein due to the fusion of the original two veins.

Such is, perhaps, as complete a picture as we can yet form of the appearance of the chorionic vesicle and its contents at this period of antenatal life. It leaves, of course, much to be desired. We know nothing of the changes taking place in the human ovum as it comes down the Fallopian tube. We know not how it develops its thick covering of epiblast cells or trophoblast. We can only suppose that soon after its arrival in the uterus it is furnished with a covering, apparently possessing all the cellular activity of an epithelioma, by means of which it eats its way into the mucosa of the uterus. While it has all the cellular activity of an epithelioma (and more), it, however, has not the unrestrained or ill-regulated mode of growth of such a tumour, for very soon an orderly development of parts begins to be evident, and a primitive placenta is evolved, while embryonic membranes, vesicles, and structures commence to appear. From the point of view of the antenatal pathologist it is much to be regretted that there is no specimen showing how, in the human subject, the amniotic sac is formed and how the umbilical vesicle arises, for these are structures which are supposed to have much to do with the origin of teratological conditions. In the earliest known human ovum both these structures are already present, and they give no indication as to their ontogenesis.

The question of the age and size of human embryos is a very difficult one. F. P. Mall's settlement of it is the best that we have at the present time. Mall (*Johns Hopkins Hosp. Bull.*, xiv. 29, 1903) has found that for embryos of from one to one hundred millimetres in length their ages can be fairly correctly estimated by multiplying the length in millimetres by one hundred, and then extracting the square root, when the result will be the age in days. The formula is $\sqrt{100 \times \text{length in mm.}}$ = age in days. For fœtuses of from 100 to 220 mm. in length, the length in millimetres equals the age in days. It is useful to remember the following leading facts, which are fairly

well established: embryos of 1 mm. are about 12 days old; of 2·5 mm., about 14 days; of 4·5 mm., about 19 days; of 7 mm., about 26 days; of 11·5 mm., about 34 days; and of 17 mm., about 41 days. After forty-one days the embryo becomes the neofœtus.

which are so complex as to baffle the keenest observer. We cannot trace out the processes in their intricacy, but we can at least recognise the results.

Through the growth of the chorionic vesicle and its contents, the decidua reflexa comes in

Fig. 9.—Guicciardi's Early Gestation Sac. Section showing—*cs.*, caduca or decidua serotina, at one pole of ovum; *cp.*, decidua vera; *cr.*, caduca or decidua reflexa; *v.*, chorionic villi; *lch.*, tract in which the chorion comes into relation with the decidua, a zone of amorphous tissue alone intervening; *ch.-ch.*, chorionic epithelium and connective tissue; *am.*, amnios; *E.*, embryo; *UV.*, umbilical vesicle. (*Vide Annali di ostetricia*, xxiv. p. 176, 1902.)

The Embryo in the Third Week after Impregnation

Several embryos of the third week are available to enable the embryologist to construct his picture of developmental events at this period; but, as it happens, there is a scarcity of material showing the state of the decidual membranes at this time. It is truly an *hebdomada mirabilis* for the embryo, this third week after impregnation, for during it there is a marvellous differentiation of organs and tissues taking place, and a transformation scene going on, the details of

contact with a larger surface of the decidua vera, and the potential space between these two membranes is diminished; for at the beginning of this third week the chorionic vesicle measures 14 mm. by 12 mm. by 5 mm. (Leopold's fifteen day specimen). Decidual cells are seen in the reflexa as well as in the serotina, and in the lower and middle part of the former there are enormously distended capillaries. In the compact layer of the serotina are likewise numerous dilated capillaries. The outer ends of the glands are very narrow, appearing as slits. In the spongy layer the gland spaces are irregular,

and contain much desquamated epithelium. Here and there are extravasations of blood. The relations of the ovum to its membranes are well seen in Guicciardi's fifteen days' specimen (Fig. 9).

The mode of attachment of the chorionic vesicle to the reflexa and serotina is such as has been already indicated. Possibly the villi are even now becoming more numerous in relation to the serotina than to the reflexa, indicating the commencement of the differentiation of the chorion into a villus-carrying part (chorion frondosum, placental part) and a portion which is bare (chorion læve, non-placental part). As yet, however, there is a circulation going on all round the chorionic vesicle (in its reflexal as well as in its serotinal part), and no indication of a limitation of it to any one area. The villi are covered with syncytium with Langhans' layer underneath, and in their mesoblastic cores capillaries can be clearly seen. In Guicciardi's specimen (*Ann. di ostet.*, xxiv. 176, 1902) only a few villi were vascularised.

With regard to the structures inside the chorionic vesicle in the third week, it has to be noted that the amnion is at first a thin membrane springing from the body of the embryo round the origin of the umbilical vesicle. It, therefore, surrounds the back, sides, and cephalic and caudal ends of the embryo, but has no relation as yet to the umbilical vesicle (or yolk-sac) and to the part of the embryo which communicates with that vesicle. It is closely applied to the embryo, there being as yet little liquor amnii. During the third week the amniotic fluid increases relatively in amount, but even at the end of the week the membrane is not far removed from the embryo, and does not surround the umbilical vesicle and scarcely covers the heart. In its microscopic characters the amnion about this period shows a differentiation: there is an epiblastic layer of somewhat endothelial cells as before, but the mesoblastic tissue now consists of an outer layer similar in appearance to the epiblast, called the mesothelium, and of an inner homogeneous matrix next to the epiblast layer.

Embryo at the Beginning of the Third Week

For our knowledge of the appearances of the human embryo, about the beginning of the third week after impregnation, we are indebted to the study of the specimens of J. Kollmann, of C. S. Minot (Nos. 195 and 143), of His (Lg, Sch. 1, and L), of A. Rondino, and of Coste. The embryo now measures from 2·2 mm. to 2·5 mm., and is still almost insignificant in size when compared with the chorionic vesicle and the decidual membranes; but it already shows a marvellous advance in the differentiation of its parts (Fig. 10). The cephalic and caudal ends are quite recognisable, and both of these project a little beyond the umbilical vesicle, which is now no longer attached to the whole length of the ventral aspect of the embryo. (In Kollmann's 2·2 mm. embryo the umbilical vesicle was attached for a distance of 1·5 mm., leaving the head to project 0·58 mm. and the tail

Fig. 10.—Embryo at the beginning of the third week. Embryo Lg. (After His.)

0·3 mm.) The cephalic end is already showing signs of enlargement, a feature which is so characteristic at later stages of embryogenesis, and is bending slightly towards the ventral aspect. There may be no branchial arches (Kollmann's embryo), or there may be two (Minot's, Nos. 143, 195) or three (Coste's). No indication of the eye or ear may be found (Kollmann's embryo); or the optic vesicles may have already grown out, and the epiblastic invagination which is to form the otocyst or auditory vesicle may have begun to form (His' Embryo Lg). The oral invagination can be seen, but the oral plate still separates it from the large primitive pharynx; below it is the first branchial arch, which by and by becomes the mandible, and below that is the first branchial slit, or rather groove, with the second or hyoidean branchial arch. The brain (vesicular in character) forms a large part of the whole embryo: there is a fore-brain from which the optic vesicles have grown out; at the junction between the fore- and mid-brain there is a sharp bend forwards (almost at a right angle) of the former part; the hind-brain is relatively very long, and near its centre lies the open invagina-

tion of the otocyst (Fig. 11). The greater part of the medullary groove, therefore, is now closed in to form the medullary canal; the part posterior to the hind-brain is not yet differentiated into the spinal cord. Between the fore-brain and the attachment of the umbilical vesicle, and lying in a large pericardial space, is the prominent heart, which consists of an inner tube formed of endothelial elements, and an outer tube of contractile elements, which ultimately form the heart muscle. This heart tube is asymmetrically bent: from the point where the great veins enter it, it runs towards the head and left side, making the auricular limbs; then it turns to the ventral side and obliquely to the right, forming the ventricular limb; and then takes a curving course to the median line, forming the aortic limb, and ends close behind the mouth. Between the two heart tubes (endocardial and myocardial) is a considerable space; and the inner or endothelial tube is continuous at one end with the walls of the veins and at the other with those of the aorta. We note, therefore, that by this date the two endothelial tubes, which represented the heart in the second week, have fused into or become one. From the aortic limb (outside the pericardial sac) arises the aorta, which divides into two branches on each side which, as the aortic arches, pass round the pharynx. The front branch on each side curves over and joins the second, to form one of the two dorsal aortæ. The dorsal aortæ pass towards the caudal end of the embryo; about midway they fuse into one dorsal aorta, which near the tail bifurcates into two vessels (the allantoic or umbilical arteries), which pass through the Bauchstiel (or allantoic stalk) to the chorion. In an earlier stage, doubtless, the dorsal aortæ pass, without fusing, to the umbilical vesicle as vitelline arteries, but of this we have insufficient evidence from human embryos. The veins which proceed to the heart consist of the cardinal, the jugular, the omphalo-mesenteric or vitelline, and the umbilical. On each side of the body the jugular vein (from the head end of the embryo) joins the cardinal vein (from the tail end) to form the ductus Cuvieri, which passes into the sinus venosus and through it into the heart. The ductus Cuvieri is joined by the umbilical or allantoic vein of the same side, which comes from the chorion by way of the Bauchstiel, and then takes, as Minot says, a short cut through the somatopleure along the base of the amnion. The omphalo-mesenteric or vitelline vein opens into the sinus venosus. There is as yet no trace of the inferior vena cava, and, since nothing more than a slight evagination or outgrowth from the fore-gut represents the liver, the veins have not the complicated hepatic relations which they afterwards develop.

With regard to the intestine or embryonic part of the archenteron at this age, it is to be noted that it is divided into three parts. Of these the fore-gut is expanded anteriorly into the wide primitive pharynx with two gill-pouches. The middle portion communicates freely with the umbilical vesicle, and shows the anlage of the liver just where it is continuous with the fore-gut. The posterior portion is distinct from the umbilical vesicle, and ends in a dilatation (His' bursa), and from the under

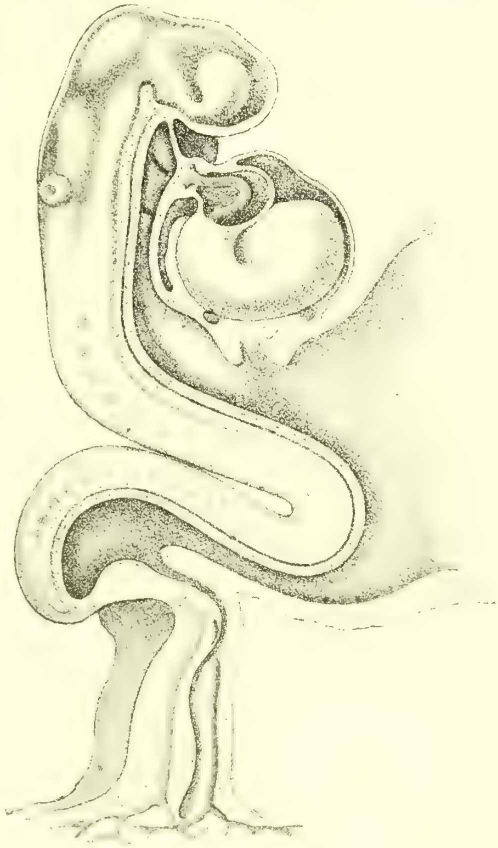


Fig. 11.—Embryo at the beginning of the third week, seen in section. Enlarged about 37 times. (After His.)

surface of this dilatation arises the allantoic diverticulum which passes into the Bauchstiel (*vide* His' Embryo Lg, Figs. 10 and 11). As already stated, the fore-gut is shut off from the oral invagination by the oral plate (Rachenhaut).

At this period (beginning of third week after impregnation) the notochord can be seen distinctly, flanked with a varying number of protovertebræ, or, to use Minot's more correct term, primitive segments. These primitive segments, which perhaps even at this age show a division into the myotome next to the medullary canal and the nephrotome (or intermediate cell mass) which lies distally, vary in number from fifteen (Kollmann's specimen) to twenty-nine (His' Embryo Lg). One of the most remarkable

features presented by embryos of the beginning of the third week is the presence of what has been called the dorsal flexure (seen well in Minot's Embryo, No. 195, and in His' Embryo Lg). By this term is meant the bending of the column of primitive segments in such a way as to form a deep dorsal concavity, possibly in order to accommodate the long back of the embryo. Minot says about this (*Human Embryology*, p. 313, 1892): "the facts indicate that the back is too long for the somatopleure at the side of the body, and that it finds room . . . by becoming concave; later it springs into a new position of equilibrium by becoming convex; it is possible that the change from the concave to the convex position is very abrupt, and it is probable that the time of its occurrence is very variable." This flexure may sometimes owe its existence to accident, but I agree with Minot that His' observations prove it to be normal in certain instances at least. From the teratologist's point of view, its presence is particularly interesting, as it is apparently maintained permanently in some monstrosities (*e.g.* in iniencephaly and exomphalos).

In order to complete our survey of the state of the embryo at this epoch, we must now glance at its caudal extremity. The prolongation of the body of the embryo by which it is attached to the chorion has now become a distinct stalk (Bauchstiel, body-stalk, allantoic stalk, later the umbilical cord). On account of the bending forwards of the caudal extremity of the embryo, the origin of the Bauchstiel likewise comes to be situated more on the ventral aspect. The stalk contains at this date the allantoic arteries and veins, and the allantoic diverticulum from the archenteron. It is appreciably nearer to the wide stalk of the umbilical vesicle, but it has not yet come into contact with it.

Embryo at the End of the Third Week

We form our visual image of the human embryo at the end of the third week after impregnation from the study of the specimens of Chiarugi, His (Embryos, Rf, M, BB, and Lr), Allen Thomson, Ecker, Hecker, von Baer, J. Müller, and R. Wagner. Organogenesis is still proceeding at a very rapid rate, for the embryo at the end of the third week differs in many particulars from the embryo at the beginning thereof; and we have not nearly enough observations of human embryos of these ages to enable us to fill up the gaps in our knowledge of the means by which one stage passes on into the next. It is undoubtedly a wonderful week (*hebdomada mirabilis*) in the history of the embryo; all the story of embryological development is wonderful, but the third week is so essentially a week of beginnings that it arrests our attention, fascinates us, and finally amazes us by the variety and complexity and rapidity of the changes which supervene upon these beginnings. When the first rudiment of an organ or part has appeared, it is not so difficult or so surprising to follow it in its progress into its mature form; but to see it emerging in the first instance out of a sort of pre-embryonic chaos, that it is which startles, astounds, and almost stupefies us. Had not one of the writers of old time a dim adumbration of all this when he wrote these words?

> Thine eyes did see mine unperfect substance,
> And in thy book were all my members written,
> Which day by day were fashioned,
> When as yet there was none of them.

To return to the description of the embryo at the end of the third week, it may be stated generally that all the systems and parts which were found indicated at the beginning of the week are now much further advanced in development, while some new parts have for the first time become evident. The umbilical vesicle is now attached by a relatively much smaller base to the ventral aspect of the body than formerly, so that we may reasonably begin to speak of the pedicle of the umbilical vesicle or yolk-stalk; the vesicle itself has become pear-shaped. It is still growing, indeed it continues to increase till the end of the fourth week, but its rate of growth is slackening. The embryo measures at this age from 2·66 mm. (His' Embryo M) to 3·2 mm. (His' Embryo BB), or even to 4·2 mm. (His' Embryo Lr). The measurements of the whole chorionic vesicle may be 7·5 mm. × 8·0 mm. (His' Embryo M), or 11 mm. × 14 mm. (His' Embryo BB), or 12 mm. × 9 mm. (Ecker's Specimen), or 12 mm. × 12 mm. × 9 mm. (Chiarugi's Specimen).

The head projects well beyond the umbilical vesicle, and shows a slight twist to left or to right; this twist is part of the spiral form which the whole embryonic body has assumed, the caudal end exhibiting a twist to the opposite side as compared with the head. There are now three (or four) branchial grooves. The mouth is relatively large; it is deeper than at the beginning of the week on account of the rupture of the oral plate; it communicates, therefore, with the fore-gut (Fig. 12). On each side of the mouth at its upper corner can be seen the maxillary process from the first branchial arch or mandibular process, which is also very evident. The pharynx is still wide, and four pouches can be easily recognised; each pouch lies between two branchial arches. A new development is the presence of a small projection on the ventral floor of the pharynx in the middle line; this is the *tuberculum impar* of His; and from it arises part of the tongue. With regard to the nervous system, it must be noted that the medullary or neural canal is now closed in its whole extent. The fore-brain, which has become quite distinct from the mid-brain, is divided into two parts, the secondary fore-brain (prosencephalon or telencephalon)

and the inter-brain (thalamencephalon or diencephalon); or rather, as Minot believes, the anlages of the cerebral hemispheres arise from the anterior end and dorsal side of the fore-brain, which, on account of the head bend which is present, grows downward and forward. The optic vesicles are by this time clearly stalked, and the otocyst is a closed pear-shaped vesicle. With regard to the primary head bend or flexure, to which reference has been made, it is to be noted that now it is so marked that no longer is the fore-brain at right angles to the hind-brain (by a flexure situated in the mid-brain), but it has become so sharply bent that its floor is almost parallel with the floor of the hind-brain. It must be remembered that the fore-, mid-, and hind-brain are not at this stage of development anything more than vesicles. At the junction of the hind-brain with the spinal cord is the medulla oblongata, and in it even at this date it can be seen that the central canal is wider in the dorsal than in the ventral portion; at this point, also, there is an indication of the second cerebral flexure or neck-bend which appears first as a projection (His' *Nackenhöcker*). In front of this region is the anlage of the cerebellum, and behind it is the spinal cord.

The heart, as seen in His' Embryo BB, is now an S-shaped tube, "the venous end is convex toward the head, the arterial end convex toward the tail" (Minot). From the arterial end spring the aortæ which give off five branches (aortic arches) on each side of the neck; these branches unite again on the dorsal side, and passing backward the resulting trunk joins that of the opposite side to form the single median dorsal aorta, which subdivides into the two umbilical or allantoic arteries. Each of the aortic arches helps with the surrounding tissue to make a visceral or branchial arch; and between the five arches are the four visceral, branchial, or gill clefts or grooves (for they are never really clefts in the human embryo). The anlages of the true auricular cavities of the heart have now appeared, and the two limbs of the ventricle are nearer together and form a distinct apex where they join. The heart area of the embryo is quite covered by amnion at the stage of development at present under consideration. The veins consist of the cardinals and jugulars, of the ducti Cuvieri formed by their union, the omphalo-mesenterics and the umbilicals; the three last named come into close relation with the anlage of the liver and also with the septum transversum, while the cardinals are anatomically associated with the developing Wolffian bodies (to which I shall immediately refer). The development of the septum transversum and of the diaphragm is a peculiarly complex and obscure part of organogenesis; but upon it the researches of Franklin P. Mall (*Johns Hopkins Hosp. Bull.*, xii. 158, 1901) and others have thrown much light. The septum transversum is, to begin with, a V-shaped bridge of mesodermal tissue (irregular round cells, numerous vessels communicating with the veins of the heart) which connects the umbilical vesicle with the embryo at the juncture of the head with the amnion. It supports the omphalomesenteric and umbilical veins, and contains in it the

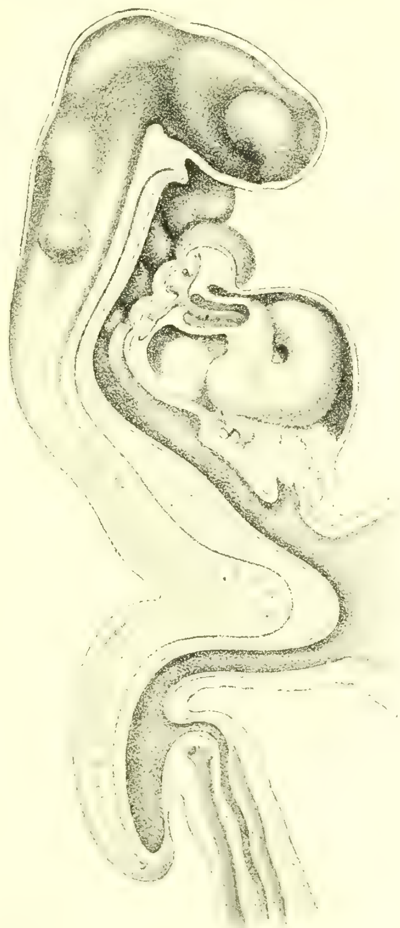


FIG. 12.—Embryo at the end of the third week. Embryo BB. Enlarged about 37 times. (After His.)

anlage of the liver as well as that of the diaphragm, and all these structures are at a high level in the body of the embryo in the neighbourhood of the structures of the head; indeed nearly the whole cœlom at this stage of development lies in the head and neck region. The septum transversum serves to divide the cœlum anterior to it or headward of it (the parietal cavity of His) from that which lies posterior (tailward) to it (the trunk cavity of His); but laterally the two cavities communicate by passages, the parietal recesses, each of which divides later into a ventral and a dorsal part. Ultimately the parietal cavity becomes the pericardial sac, while the trunk cavity with the

dorsal parietal recesses becomes the pleuro-peritoneal cavity. By the end of the third week the liver diverticulum, now enlarged and branching, is found embedded in the septum transversum. Through the descent of the septum all these associated parts greatly change their relations to the framework of the body.

At this date, also, traces of the Wolffian bodies can be recognised as little canals in the Wolffian ridges; the latter are longitudinal ridges, one on each side of the dorsal surface of the cœlom of the abdomen. The tubules of the Wolffian body (mesonephros) are evidently developed from the nephrotomes or intermediate cell masses of the primitive segments of the neighbourhood. The blood from these bodies passes to the cardinal veins. Thirty or more primitive segments or protovertebræ can be recognised (His' Embryos BB and Lr), and the dorsal flexure has now disappeared, a convexity taking the place of the concavity which was present at the beginning of the third week. A tendency is present for the embryo to bend forwards, the cephalic and caudal extremities approximating, and this tendency becomes more evident in the fourth week. The curving forward of the caudal end of the body brings the insertion of the Bauchstiel still more to the front, and so it and the umbilical vesicle lie nearer to each other than formerly.

Recapitulation

Thus, in these two weeks, we see the embryo developing from a mere disc of tissue resting upon a relatively large umbilical vesicle into an organism with a circulatory apparatus, a series of vesicles constituting a nervous system, an intestinal canal, a body cavity with a commencing septum dividing it into a thoracic and an abdominal portion, a mouth consisting of a five-sided fossa, a pharynx with branchial arches and clefts, a series of mesodermic somites or primitive segments, and rudiments of eyes, ears, and Wolffian bodies. This organism no longer rests, insignificant in size, upon its umbilical vesicle, but is of relatively considerable size, and has its umbilical vesicle appended to it by a recognisable vitelline stalk. All these structures have appeared during these eventful weeks; and they have come into being by a series of changes of which we know very little from the direct examination of human embryos, but about which embryologists have formed many theories from the observation of the early stages of development in the lower animals. We cannot, in consequence, be at all sure of the manner in which many of the transformations and alterations have been produced. We know nothing, for instance, of the way in which the neurenteric canal is closed, and but little of the relation of the allantoic stalk to the Bauchstiel. In whichever direction we attempt to follow out the problems of the subject, we immediately feel the pressure of the limitations of our knowledge. At the same time the feeling of restriction thus produced must not be suffered to engender in us the idea that we are disqualified from ultimately searching out the secrets of organogenesis. When we think of the progress that has been made in the last twenty years, when we reflect how much the finding of such a specimen as Peters' early ovum may mean for the elucidation of our problems, and when we remember the hundreds of skilled workers engaged in this field of research, we take courage again and press forward. Every advance, it is true, but adds to our perception of the regions yet unknown; but this was to be expected, for the domain of Embryology is of vast extent. We are not, therefore, to be hindered but stimulated by the prospect ever opening up before us; the wider view is in itself a gain. Let us then go on carefully preserving early embryos, cutting them in serial sections, and reconstructing them again, so as to obtain an intimate knowledge of their inmost constitution. Scarcely any two will be found perfectly alike, and we may at any time make a discovery rivalling that of the detection of the neurenteric canal in the human embryo. Let us also examine all uteri and appendages removed by hysterectomy, vaginal or abdominal; such operations are common nowadays; we may thus add to the number of the known specimens of early ova *in situ* in the uterus, we may even find an impregnated ovum in the tube on its way thither.

The Embryo in the Fourth Week

In the previous section an attempt was made to place in graphic fashion before the reader's mind a picture of the conditions found inside the uterus during the second and third weeks after impregnation. The picture was confessedly an imperfect one: many details were dimly represented because imperfectly known; others were only, so to say, outlined in the rough; while others were altogether wanting. Yet, after all, some idea was gained of the nature of the processes of organogenesis going on at this period of antenatal life. Let us now, in this section, try to do at least as much for the fourth week of embryonic existence. The attempt is likely to be more successful, for the number of known and examined specimens of embryos of this period is much larger than at the earlier dates. Let us pursue the same plan and consider first the extra-embryonic parts of the gestation sac and then the embryo itself.

Extra-embryonic Parts in the Fourth Week

Regarding the decidual membranes at this period of intra-uterine life we gain some information from the description given by E. Fraenkel (*Arch. f. Gynæk.*, xlvii. 139, 1894) of an aborted ovum, three and a half weeks old, and containing

an embryo which measured 4·3 mm. in length and possessed thirty pairs of primitive segments. The sac formed by the decidua reflexa (*capsularis s. circumflexa*) measured 16 mm. in width at its base by 10 mm. in height. Two parts of the reflexa could be easily distinguished: a thin polar portion with no regular structure, with scarcely any traces of glands, and with signs of commencing degeneration; and a thicker basal portion, the inner half of which was more compact (no blood sinuses and very few glands), while the outer half, especially near its junction with the decidua vera, showed several gland orifices and many blood sinuses. The decidual cells were more compressed in the inner than in the outer half. In the decidua serotina the large decidual cells were practically limited to the compact layer. Blood sinuses could be recognised opening into intervillous spaces both in connection with the serotina and the reflexa. C. Kupffer's specimen, of about the same age, showed similar conditions (*München. med. Wchnschr.*, xxxv. 515, 1888); in this case also villi were found over the whole surface of the chorion although they were most strongly developed in an equatorial band. An epithelial double layer covered the chorion and the chorionic villi (Fraenkel's specimen), the outer layer being, as at an earlier date, plasmodial or syncytial, and the inner cellular. In the neighbourhood of the polar portion of the decidua reflexa the syncytium was very thick and was covered with a blood coagulum; in this spot the cellular layer was very thin. The stroma of the chorion reached its greatest thickness opposite to the decidua serotina. The villi were most numerous where the decidua serotina passed into the decidua reflexa. Kastschenko (*Arch. f. Anat. u. Entwcklngsgesch.*, p. 451, 1885) has described a fine network of threads in the syncytial layer at this age of pregnancy, and he found the syncytium thickest over the ends of the villi, while buds and strands extended from their sides in the form of processes. The amniotic membrane in the fourth week has very similar appearances to those described already (in the preceding section) as existing at the third week. The amniotic sac, on account of the rapid growth of the embryo, is at this time almost entirely filled by it, so that the amnion is closely applied to the embryo. This space between the amniotic sac and the wall of the chorionic sac, the extra-embryonic cœlom, is still large.

The umbilical vesicle or yolk-sac is now quite extra-embryonic in position, and is attached to the body by a more constricted pedicle, at first short and later of a considerable length. Towards the end of this week the Bauchstiel or allantois-stalk has grown partly round the proximal part of the yolk-stalk, so that we can now speak of the umbilical cord, although it is not yet very cord-like. The umbilical vesicle, in this week, reaches its greatest development; it measures from 2·7 by 3·0 mm. (His' embryo *a*) to 5·0 by 7·0 mm. (Mall's embryo of 26 days). The umbilical vesicle can be recognised as differentiated from the yolk-stalk, and the cavity in the latter (vitelline duct) is soon obliterated.

The Embryo in the Fourth Week

For the study of human embryos of the fourth week (22 to 28 days) several specimens are available, among which may be named His' embryos *a*, D_2, W, R, B, A, Eck 1, and Stt, and those of C. Rabl, Coste, Allen Thomson (fourth embryo), Hensen, Ecker, Fol, B. H. Buxton, Mall, J. Müller, V. Magnus, and Waldeyer.

The embryo of this age measures from 5 to 7·5 mm. in length; but the organism is now so coiled upon itself that it is difficult to ascertain its real length. The dorsal flexure of the preceding week has entirely disappeared and is replaced by a well-marked and rounded convexity posteriorly, which is due to the approximation on their ventral aspect of the caudal and cephalic ends. The embryo is shaped like a C, the upper end of the C being the fore-brain and the lower end the embryonic tail; or, rather, the organism is so rolled up that its dorsal outline describes more than a complete circle (Fig. 13). In addition to the head-bend (in the region of the mid-brain), there is also a well-marked cervical flexure or neck-bend (at the posterior limit of the hind-brain); and the well-developed tail is sharply bent forwards at the sacral bend. So it comes about that the length of the embryo from the head-end to the tail-end does not represent the total extent of the organism, thus His' embryo *a* measured 4 mm. but when unrolled 13·7 mm.

The body shows still the spiral twist which was noted at the end of the third week: the head is bent to the right and the tail to the left in His' embryo *a*. The Bauchstiel or allantoic stalk is now becoming more stalk-like; on account of the marked ventral concavity of the trunk and the development of the tail, it lies near the yolk-stalk and partly surrounds it, as has been already pointed out.

A very noticeable change in the external appearance of the embryo is now visible, namely, the indication of the limb-buds. Possibly they may be recognised in some specimens of the end of the third week; at any rate, they are quite distinct at the beginning of the fourth. They are at present no more than slight swellings or flat processes arising from the ridge (Wolffian ridge) which runs along the dorsal border of the embryonic somatopleure. In structure they consist of rounded mesoblastic cells with an external layer of epiblast. Each limb-bud has an upper, dorsal, or extensor surface, and a lower, ventral, or flexor one; and there are two borders,

an anterior or cephalic, and a posterior or caudal. Into these limb-buds grow processes from the myotomes of a number (not less than seven) of the primitive segments. The extremities are at this time truly appendicular *buds* and not *limbs*, for they are quite unsegmented. The anterior ones are larger than the posterior. The limbs, as may be pointed out, belong to the outlying parts of the developing organism and so are late in showing differentiation ; thus it comes about that in the fourth week, when other systems are far advanced in development, the extremities are only little mesoblastic outgrowths.

The embryo is still somewhat translucent, so that the internal parts can be dimly discerned. Thus it can be seen that there are at least twenty-seven primitive segments or somites (Mall's 26 day embryo, Figs. 14, 15, 16), and there may be thirty or more (cervical 8, dorsal 12, lumbar 5, sacral 5). There were thirty-five in Magnus' specimen. In the region of the head the outlines of the five chief divisions of the brain can be recognised, and the margins of the fossa rhomboidalis (future fourth ventricle) are distinct; the ganglia of some of the cranial nerves are also visible, and to them reference will be made below.

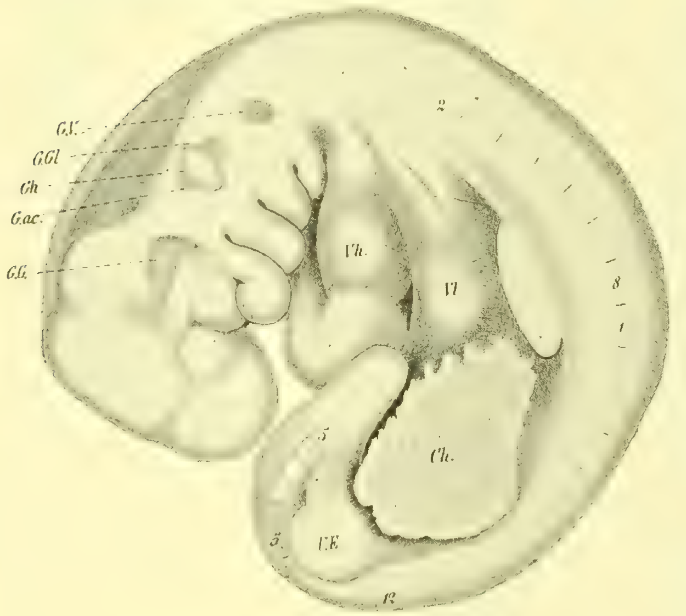


FIG. 13.—Embryo of the fourth week. Embryo *a* (after His). Enlarged 20 times. *G.V.*, vagus ganglion; *G.Gl.*, ganglion of glosso-pharyngeal; *Gh.*, otic vesicle; *G.ac.*, auditory ganglion; *G.G.*, Gasserian ganglion; *Vh.*, heart; *Vl.*, liver; *U.E.*, lower extremity; *Ch.*, chorion.

Three or four branchial grooves and arches can be made out during this week ; they have irregular outlines, and are beginning to be transformed into the more permanent arrangement of parts. Towards the end of the week the sinus præcervicalis is well marked, and the fourth branchial arch lies deeply in it, and largely under cover of the third arch ; the third arch, also, is within the sinus. This cervical sinus is formed by the growth of the second arch over the third and fourth arches till its tissues come into contact with the body wall beyond them in the region of the faintly marked fifth arch ; thus the epiblast of these arches and clefts is buried. This change, however, is only beginning in the fourth week. From the dorsal or attached end of the first branchial or mandibular arch a projection can be seen arising ; this is the superior maxillary process, and it can be recognised both in the beginning and towards the end of the fourth week (*e.g.* in His' embryo *a*, and in Mall's 26 day embryo). The ventral or free end of the first arch is knob-like, so also is that of the second or hyoid arch ; in fishes it forms the operculum for the gills, while in the human embryo, as above stated, it serves to close in the sinus præcervicalis. It is to be noted that neither now nor at any other time do the branchial furrows or grooves in the human subject become continuous with the pharyngeal pouches ; the epiblast comes into touch with the hypoblast (no mesoblast intervening) to form the "closing membrane," but there is no communication between the external branchial clefts and the internal pharyngeal pouches.

During this week another change in the external appearance of the facial region of the embryo occurs, the appearance of the first indications of a nose. On each side of the naso-frontal process which grows down between the two superior maxillary processes is an epiblastic thickening, the olfactory plate (as in His' embryo *a*) ; these two olfactory plates, after the twenty-fifth day, become hollowed out (or, rather, their margins grow up) to form the nasal pits (as in His' embryo *a* or in Mall's 26 day embryo). The pits, however, remain shallow during this week ; further, their margins do not grow up on the side next the mouth, and so they open freely into the oral cavity. From the nasal pits the nasal mucous membrane ultimately develops. Just outside and anterior to the nasal pits are situated the optic vesicles which are now stalked and converted into optic cups ; and each shows (at the end of this fourth week) a lens surrounded by a groove which also passes down between the superior maxillary process and the nasal pit of the same side. The lens is formed by the thickening of the epiblast at the point where the optic vesicle comes nearest to the surface. The otocyst or otic vesicle (formed from the auditory pit in the third week) has

now receded from the surface of the epiblast or ectoderm; it becomes pear-shaped rather than spherical, and forms a labyrinth in which vestibular and cochlear portions and a recessus vestibuli can be dimly recognised.

Such are the more striking and evident external features of the embryo of the fourth week. We must now try to form a picture in our mind of the internal arrangements found at this epoch, and it will be convenient to study in order the nervous, alimentary, circulatory, urinary, and supporting systems of the embryonic body.

The spinal cord is the less differentiated part of the nervous system at this age, so it will be convenient to study it first. It consists of a tube with a central cavity which is still relatively large and has an elongated oval form, the long diameter of the oval being antero-posterior. The lateral walls are thick; the anterior and posterior are thin, and are called the roof- and floor-plates or mid-dorsal and mid-ventral laminæ. At the beginning of the fourth week two kinds of tissue-elements can be made out in the neural tube wall: spongioblasts or sustentacular elements and neuroblasts or young nerve-cells (the future neurons). To begin with, the wall consists of a single layer of columnar cells or spongioblasts, but soon at the point where these cells border the lumen of the canal other elements appear between them called germinating or germinal cells, and from these arise the neuroblasts. The spongioblasts multiply and form the sustentative or sustentacular part of the spinal cord, and more particularly what is called the neuroglia. The inner ends of the spongioblasts unite to form a close network next the canal cavity, the inner limiting membrane or ependymal layer. There is an outer layer formed by the branched prolongations of the ependyma cells, which forms the marginal velum or Randschleier of His. Between these two layers is a very delicate network, which becomes the mantle layer, and into its meshes the neuroblasts wander. This wandering would seem to take place by an accumulation of the protoplasm on the distal

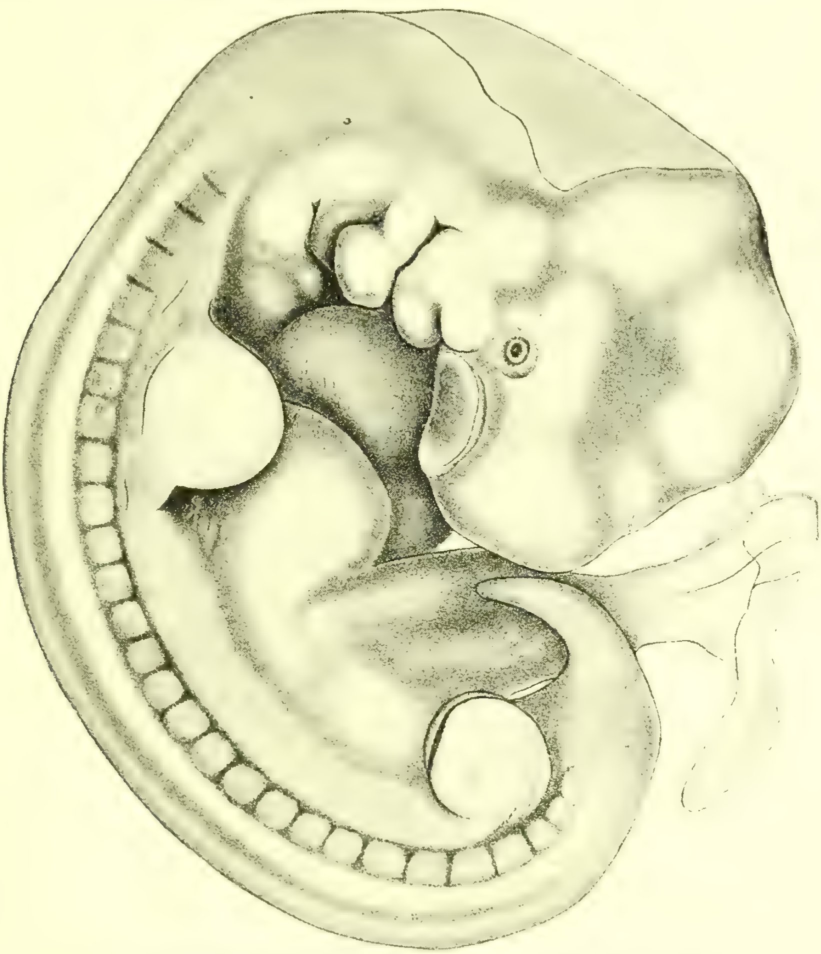


FIG. 14.—External appearances of Mall's 26 day embryo. Enlarged about 18 times. (*Vide* Mall's article in the *Journal of Morphology*, vol. v., 1891.)

side of the nucleus, and this indicates the commencement of the formation of the axis-cylinder process or axon of the future neuron. Later, other processes arise from the neuroblast, the dendrites. The axons pass out through the marginal velum, and some form the ventral nerve-roots.

While these changes have been going on inside the spinal cord the cells of the neural ridge or crest (line of union of the neural tube) begin to form the posterior or dorsal root ganglia; these cells arrange themselves segmentally at the sides of the cord, some of them (neuroblasts) take on a fusiform shape with a process extending from each end of the spindle, and one of these processes (the axon) grows inward, penetrating the marginal velum, to come into contact with the dendrites of the neuroblasts of the mantle layer. Thus the two nerve-roots of the spinal nerves are developed, the ventral ones by cell processes growing outward, the dorsal ones by processes growing inward. Thus, also, a beginning is made with the differentiation of the cord into a grey and a white part, but as yet there is no contrast in colour, for the medullary sheaths of the nerves do not appear till the fifth month of antenatal life.

So far has the development of the cord progressed by the end of the fourth week that twenty-nine spinal nerves (cervical 8, dorsal 12, lumbar 5, and sacral 4) can be counted (Mall's 26 day embryo), each with a large dorsal ganglion and a ventral root. The ganglia are largest in the cervical region, and the eight cervical nerves are united by anastomoses, the future cervical and brachial plexuses. Posterior to the fourth sacral nerve, the dorsal ganglia are not fully separated from the spinal cord.

During this (fourth) week there are as yet no sympathetic ganglia to be recognised; but, from the first six dorsal nerves, branches extend towards the chorda dorsalis; and three of these lie in front of the cœliac axis and three behind it. From these branches arise the solar plexus and the splanchnic; at any rate this view commends itself to embryologists, for it brings the sympathetic into line with the cerebro-spinal system as derivatives of the epiblast or ectoderm. During development the cœliac axis comes to lie nearer the caudal extremity, and so the sympathetic twigs which surround it also lengthen.

The brain during the fourth week shows a marked increase in complexity, and requires careful study. Five parts of the neural canal in the cephalic end of the embryo can now be recognised: there is first the end-brain, telencephalon, or hemisphere-brain; behind it (above it topographically) is the inter-brain, 'tween-brain, diencephalon or thalamencephalon; behind it is the mid-brain or mesencephalon; and then follows the hind-brain or rhombencephalon, consisting of the two parts, the secondary hind-brain or metencephalon, and the after-brain or myelencephalon. Each of these parts contains a cavity. At the beginning of the fourth week the cavity of the telencephalon is single and communicates by a wide opening with the diencephalon; but towards the end of the week the hemisphere-brain is formed by two oval projections whose cavities are the future lateral ventricles, and they communicate with the rest of the cavity of the telencephalon, which, together with that of the diencephalon, forms the third ventricle. The openings between the lateral ventricles and the third are at this time very wide; later they become the foramina of Monro. There is a thin strip of tissue between the foramina of Monro, which is really the anterior end of the neural tube; it is called the lamina terminalis. The cavity of the hind-brain or rhombencephalon is represented in after-development by the fourth ventricle; and the cavity of the mesencephalon, although now capacious, becomes the narrow iter, or aqueduct of Sylvius, of later dates. The mesencephalon becomes continuous with the metencephalon in a constricted portion, named the isthmus rhombencephali. Three flexures can now be seen in this region: the cephalic flexure in the neighbourhood of the mesencephalon, already noted and described in the preceding section; the cervical or nuchal bend, of which only an indication existed at the third week; and the pontine flexure in the region of the metencephalon, which is a flexure simply of the ventral side of the neural tube, and does not involve the whole head as the other two bends do. In the fourth week the cephalic and cervical bends are very marked, but the pontine one is only beginning to be indicated.

The walls of the neural tube in the region of the brain vary in thickness. Those of the mesencephalon and rhombencephalon are thicker than the parts anterior to them. In the myelencephalon the ventral wall alone thickens, the dorsal remaining very thin. The walls have the same general arrangement as in the region of the spinal cord: there is a roof-plate and a floor-plate, and between them are the thicker lateral walls which soon show the indications of a subdivision into a dorsal part or alar lamina, and a ventral part or basal lamina. These fundamental divisions of the neural tube are, before long, lost sight of in the great complexity of parts produced in the region of the brain; but they are there, although hidden, and have much to do with the architecture of this all-important part of the body.

The ganglia of some of the cranial nerves can now be recognised. At the beginning of the fourth week traces of the ganglia of the trigeminal or fifth nerve, of the auditory-facial, of the glosso-pharyngeal or ninth, and of the vagus or pneumogastric can be identified,

although not very clearly; but by the end of the week their positions and relations, along with those of other of the cranial nerve ganglia, can be definitely ascertained. To take the nerves in their order, it has to be noted that the cells on the concave (oral) side of the nasal pit are pyramidal in shape, and have their bases directed towards the outside of the body; they indication of the origin of the third or oculomotor cranial nerve is found in the floor (basal lamina) of the mesencephalon; while there is a well-marked group of cells in the ventral wall (basal lamina) of the isthmus rhombencephali, from which the fourth or trochlear nerve arises.

The Gasserian (or semilunar) ganglion of the fifth or trigeminal nerve is quite recognisable,

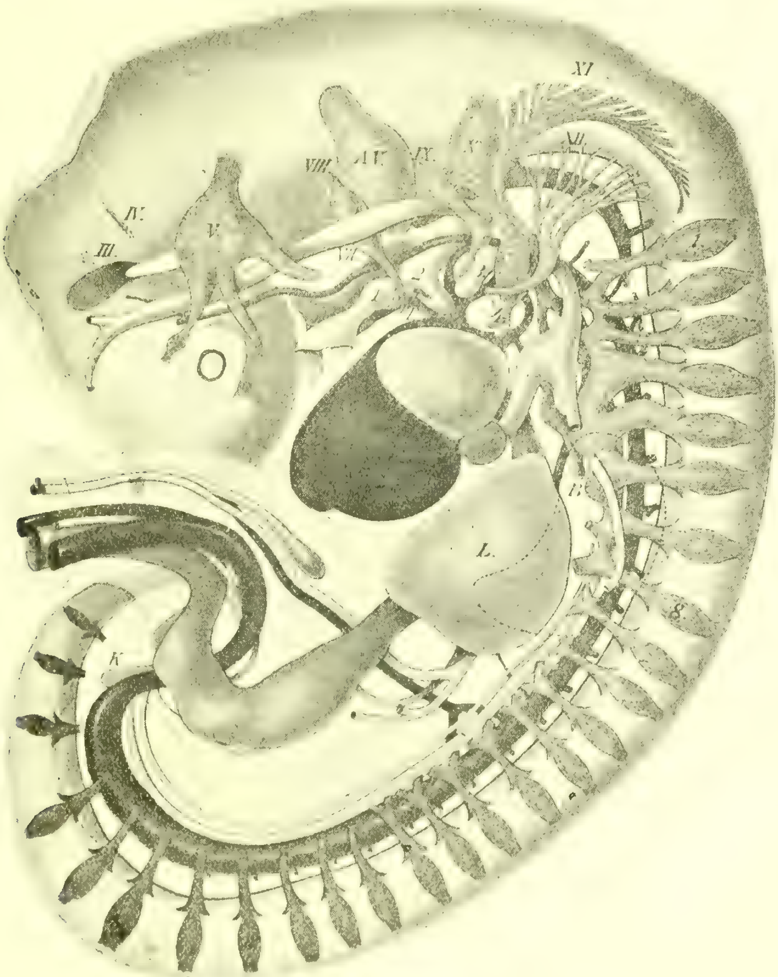


FIG. 15.—Reconstruction of Mall's 26 day embryo. Enlarged about 18 times. *III.*, *IV.*, *V.*, etc., cranial nerves; *A.V.*, auditory vesicle; 1, 2, 3, and 4, branchial pockets; *T.*, thyroid gland; *B.*, bronchus; *L.*, liver; *K.*, kidney. The dotted lines mark the extremities; the spinal nerves and the blood-vessels are indicated. (*Vide* Mall's article in the *Journal of Morphology*, vol. v., 1891.)

may be regarded as an indication of the beginning of the olfactory or first nerve. There is as yet no trace of an optic nerve; the primary optic vesicle is still in communication with the cavity of the fore-brain, and in the secondary optic vesicle (optic cup) two layers of cells can be recognised—a proximal or pigment layer, and a distal or rods-and-cones layer (with a hyaline and a cellular zone). Even at this date a vessel, the arteria centralis retinæ, can be seen (Mall's 26 day embryo); it perforates the hyaline zone of the layer of future rods and cones. A possible as is also a small group of cells upon its ophthalmic branch which constitutes the ciliary ganglion. Fibres forming the large sensory root (dorsal) pass from the ganglion into the hind-brain; the motor root arises more ventrally from the basal lamina of the hind-brain, and passes into the inferior maxillary division of the nerve. A group of cells in the basal lamina of the upper part of the hind-brain represents the sixth or abducent nerve. It is difficult at this age to separate the ganglia of the auditory (eighth) and facial (seventh) nerves; both these

nerves arise from the alar lamina of the hind-brain. The acoustic ganglion is adherent to the auditory vesicle, which is pear-shaped, and has a prolongation from its dorsal side, which indicates the beginning of the aqueductus vestibuli. Between the auditory vesicle and the myelencephalon or after-brain lies the upper ganglion of the glosso-pharyngeal or ninth nerve, which arises from cells in the alar lamina; on the ventral side is a second ganglion, the ganglion petrosum. The vagus nerve is represented by two large ganglia, the future ganglion nodosum and ganglion jugulare; these are united by a band of cells, and from the ganglion nodosum a branch passes to the fourth branchial arch. The spinal accessory arises in a row of bundles which lies between the vagus and the first cervical nerve, and emerges from the myelencephalon midway between its dorsal and ventral walls. In Mall's 26 day embryo (upon which this description is largely based) there was no trace of the rudimentary ganglion of the accessory, which has been found by His in the human subject; this is perhaps to be explained by the fact that it is an organ in process of degeneration, and so may be expected to be occasionally absent. The hypoglossal or twelfth nerve arises from a group of fibres parallel to but more ventral than those of the spinal accessory. It is evident, therefore, that already at the fourth week very considerable progress has been made with the ontogenesis of the complicated part of the mechanism of the nervous system known as the cranial nerves. It may be noted in passing that the trigeminus may be regarded as the nerve of the first or mandibular branchial arch and groove; the facial as that of the second or hyoid arch and of the

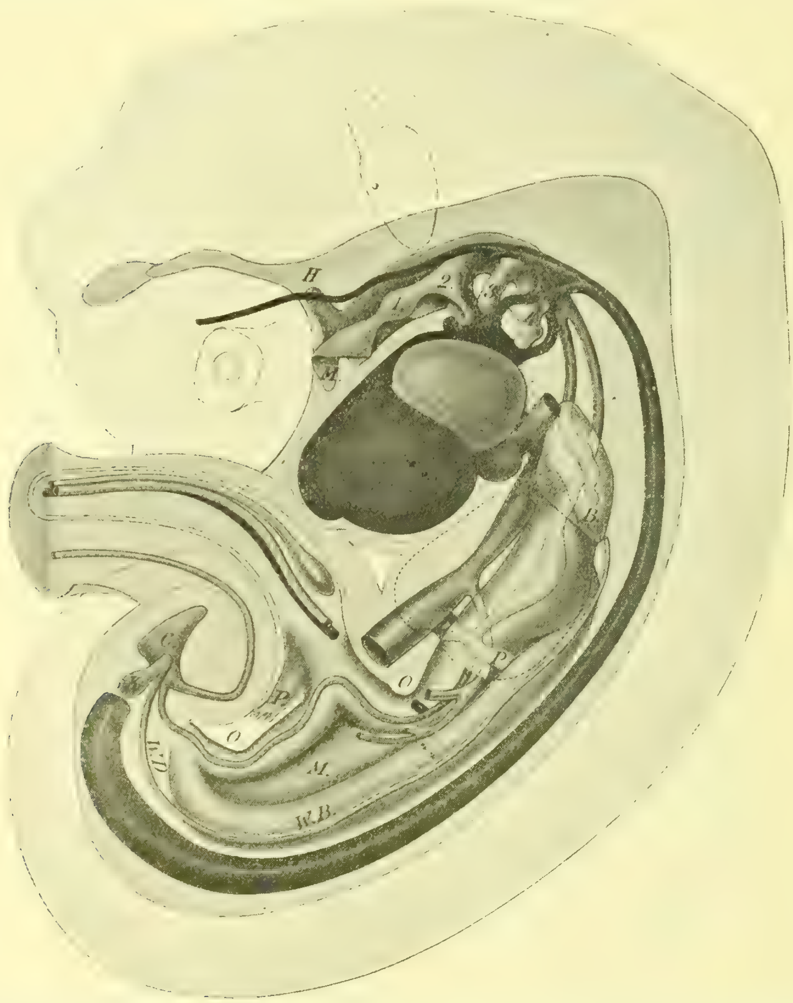


FIG. 16.—Deeper view of reconstruction of Mall's 26 day embryo. Enlarged about 18 times. *H.*, hypophysis; *M.*, mouth, mesentery; 1, 2, 3, and 4, branchial pockets; *B.*, bronchus; *P.*, pancreas; *L.*, liver; *W.B.*, Wolffian body; *W.D.*, Wolffian duct; *K.*, kidney; *C.*, cloaca; *O.*, openings by which the pleuro-peritoneal cavities communicate; *P.*, papilliform projection into the lower opening. (*Vide* Mall's article in *Journal of Morphology*, vol. v., 1891.)

groove in front of it; the glosso-pharyngeal as that of the third or thyro-hyoid arch and groove in front of it; and the superior and inferior laryngeal branches of the vagus as the nerves of the fourth and fifth branchial arches respectively.

Like the nervous system, the alimentary tract has likewise been increasing in complexity during the third week The mouth cavity is still not clearly delimited from the pharynx. The *tuberculum impar* which becomes the buccal part of the tongue was noted in the third week; it arises from the first or mandibular branchial arch, and from the first interbranchial space; it now increases in size, and is marked off by the two alveolo-lingual grooves; and behind it is a V-shaped groove with a deep depression at its apex, the future foramen cæcum. The pharyngeal or posterior part of the tongue is formed from a thickening of the ventral ends of the second branchial arches, and into the angle between them the buccal part, so to say, fits. Behind the *anlage*[1] of the tongue is an inverted U-shaped ridge, the furcula, derived probably from the ventral parts of the third or fourth branchial arches, which later becomes the epiglottis. Behind it, at the end of the fourth week, is the narrow opening into the pulmonary diverticulum from the ventral aspect of the pharynx; from this diverticulum two lung buds have been evaginated, and these push their way into the isthmus of the cœlom (the neck of communication between the pericardial and peritoneal sacs), and form the pleural parts of the cœlom. The median part of the diverticulum forms the larynx and trachea. Rathke's pouch, an invagination found in the roof of the oro-pharyngeal cavity, has not yet come into contact with the infundibular prolongation of the telencephalon to form the hypophysis; this occurs in the fifth week. In the depression (foramen cæcum) behind the tuberculum impar, a bilobed body develops, which later constitutes the median lobe of the thyroid gland, and takes up a position lower in the neck. Thickenings of the tissues in the posterior and anterior walls of the fourth and third branchial furrows develop later into the lateral thyroids and the parathyroids; they are scarcely recognisable even as thickenings at this age.

The œsophagus is comparatively short on account of the high level of the diaphragm (septum transversum) at this age. The intestinal tube below the œsophagus is dilated slightly in spindle fashion to form the *anlage* of the stomach. In connection with the duodenum the *anlage* of the liver has now taken the form of two lobes, a right and a left; and it is said that the hepatic cylinders (future bile-ducts) possess a lumen. Another bud from the duodenum is recognisable at the end of this week: it is the small group of cells constituting the *anlage* of the pancreas, and it lies in the mesogastrium (the future great omentum). Below this level the intestine makes a loop toward the ventral aspect, and to this the yolk-stalk or vitelline duct is attached. The part of the loop above the vitelline duct, along with a portion of the part below, becomes the small intestine; the rest of the tube forms the large intestine, which at this age is chiefly constituted by the entodermal cloaca. The cloaca has a pyramidal form, the apex pointing tailward; there is, through the presence of the cloacal membrane, no opening of the cloaca on the surface of the body. The small tube known as the allantois forms a projection from the entodermal cloaca; and on each side of it and dorsally is the opening of a Wolffian duct. It is to be noted that the entodermal cloaca does not form the extreme caudal part of the intestine; that is constituted by the end-gut, which begins to wither during the fourth week. For the end-gut Berry Hart has proposed the name Pars Ultima, and for the entodermal cloaca that of Pars Penultima (*Trans. Edin. Obst. Soc.*, xxvi. 259, 1901), and the nomeclature is concise and descriptive. About the end of the fourth week, or a little later, the penultimate part (entodermal cloaca) shows signs of division into an anterior and a posterior cavity by a coronal septum; the anterior cavity forms the bladder (according to the view of Keibel and others, strongly supported by Berry Hart, *Brit. Med. Journ.*, ii. for 1902, p. 773), the allantois only playing a subordinate part in its formation. The rest of the anterior cavity constitutes the urino-genital sinus, and the posterior part develops into the rectum.

The consideration of the urinary organs at this date of embryonic life may be suitably taken up here in connection with the description of the entodermal cloaca, for, as has been stated, the Wolffian ducts open into this part of the primitive intestine. The Wolffian duct extends throughout the Wolffian body or mesonephros, on the dorsal side of it in the lumbar region, and on the ventral in the cervical. The Wolffian bodies are large lobulated structures, situated one on each side of the intestine from the cloaca to the sixth cervical nerve; glomeruli can be recognised in them from the sixth cervical to the fifth lumbar nerve, and there are about three glomeruli to a segment (in Mall's 26 day embryo). Just before the Wolffian duct enters the cloaca, a small blind tube arises from it; in process of time this is converted into the ureter and kidney (metanephros). This blind tube appears during the fourth week; it is not present at the beginning of it (*e.g.* in His' embryo *a*).

The circulatory apparatus, like the other systems of organs, shows marked developmental advances during this week (the fourth) of intra-uterine life. The separation of the single atrium

[1] Anlage, meaning the first outline or indication of a part or organ, is a useful German word with no exact English equivalent.

into two auricles by means of a ridge growing from its dorsal and cephalic walls begins at this date; the separation, however, is incomplete, the opening between the auricles constituting the foramen ovale, or rather the ostium primum. The right auricle is larger than the left, and into it (the former) opens the sinus venosus or sinus reuniens, into which in turn open the vitelline and umbilical (allantoic) veins and the ducts of Cuvier. The opening of the sinus reuniens is guarded by a valve. The common atrio-ventricular canal is now divided by an incomplete septum into two auriculo-ventricular openings, by means of which the right and left auricular cavities communicate with the right and left ventricular cavities respectively, for by this time the common ventricular chamber is partly divided into two cavities. The two ventricular cavities, however, still freely communicate with each other, for the ridge or septum on the inside which corresponds to the sulcus inter-ventricularis on the outside does not yet completely divide the ventricular space. The truncus or bulbus arteriosus is by the end of the fourth week nearly completely divided into two tubes. Already the symmetrical arrangement of the five aortic arches has broken down and been replaced by a new order. The third, fourth, and fifth arches of both sides persist, and these unite to form the two aortæ, which in their turn unite, between the sixth and seventh cervical nerves (in Mall's 26 day embryo), to form the single aorta. The aorta is much larger in the dorsal and lumbar regions than in the cervical, and in the lumbar region it divides abruptly into the two umbilical (allantoic) arteries. The pulmonary artery on each side can be seen arising from the fifth aortic arch as a comparatively small branch. The first and second aortic arches have by the end of this week (the fourth) apparently disappeared. The internal carotid artery with some branches can be recognised arising from the third arch. On each side of the dorsal aorta are (in Mall's 26 day embryo) twenty-one segmental arteries, the first being in front of the first cervical nerve, and the last behind the twelfth dorsal. The second segmental artery communicates with the vertebral, a large branch which extends forward to the region of the otic vesicle and gives off two twigs, the future anterior and posterior cerebellar arteries. The subclavians grow out from the dorsal aortæ, or from the segmentals opposite the fourth arches. Between the lateral and ventral sides of the aorta there are fourteen pairs of segmental branches which supply the Wolffian bodies. From the ventral side of the aorta the cœliac axis and the omphalo-mesenteric artery arise. Through the growth tailwards of the parts in the neck, including the heart and diaphragm, the origins of all these vessels are constantly moving backwards, as it were, in relation to the segments of the body.

The same general arrangement of the veins persists, with the exception that the left or sometimes the right omphalo-mesenteric or vitelline vein has now disappeared; the remaining vein (as portal vein) unites with the umbilical vein in the region of the liver, but before this happens it receives an inferior mesenteric branch. The liver is developed around the umbilical and omphalo-mesenteric veins. The cardinal vein (which receives the blood from the Wolffian body) joins with the jugular of the same side to form the ductus Cuvieri which opens into the sinus reuniens. Veins from the upper limb-bud (subclavian) also open into the duct of Cuvier or into the jugular vein. The jugular and cardinal veins also receive segmental branches. The right umbilical vein has usually disappeared by this time.

With regard to the supporting tissues of the embryo in the fourth week certain facts are well established. The notochord is still recognisable, and stretches from the caudal extremity to a point below the hind-brain. On each side of it are the mesoblastic segments (consisting of myotomes, sclerotomes, and cutis plates or dermatomes), and in its anterior part the vertebral arteries are also situated laterally. It is usually stated that cartilage does not begin to form in the sclerotomes till the second month, but Mall describes it already in his 26 day embryo. These sclerotomes grow round the notochord, and are transformed at a later date into the spinal column, the basi-occiput, and part of the basi-sphenoid. From the myotomes are formed the muscles of the body and limbs.

Recapitulation

Like the weeks that precede it, the fourth is full of organogenetic activities. Construction is rapidly going on; it is true we have not to chronicle developments so astonishing as those of the third week, that *hebdomada mirabilis* to which reference has lately been made, yet those we have to deal with are wonderful enough, and in all respects more than difficult enough to explain or attempt to explain. We see the embryo beginning to assert itself even in the matter of bulk, while the umbilical vesicle has already reached its maximum size, and has more than reached its maximum importance. The curious dorsal flexure of the embryo has suddenly disappeared, and our visual image of the new organism now is a C instead of an inverted S; this C shape the embryo never quite loses again, and in the fœtus we recognise it in the familiar attitude of flexion; in a sense, there is even a reminiscent and instinctive assumption of it by the adult when in fear of bodily injury or hurt.

In this fourth week, also, there are seen for the first time the indications of limbs. They are nothing more than buds, it is true; but their advent is very striking, for we now realise

what it was that gave to the limbless embyro part at least of its grotesque and non-human appearance. The embryo is still far from human in its aspect, the presence of the branchial arches and grooves quite prevents that. Not yet have these gill-like structures begun to disappear, buried from sight, in the cervical sinus; that fate awaits them in the next week. The first trace of a nose is now visible in the very unnose-like nasal pits, too shallow almost to deserve the name of pits; stalked vesicles represent the eyes; and the otocysts, by their retreat from the surface, have for the time removed even the suggestion of ears.

It is difficult to recognise the simple neural tube of the early days of development in the complicated series of brain vesicles of the fourth week; and yet the nervous system is only beginning to assume its labyrinthine form with its sinuous or tortuous passages, with the intricate ramifications of its cavities, and with its endless irregularities of contour, its invaginations and evaginations. We can still identify the central cavity of its various parts, for it is throughout fairly wide and canal-like, but soon this character disappears, and it is difficult to see in the narrow iter of later weeks the wide tract of the mesencephalon of the fourth week. Already the lateral walls are thickening irregularly, while the roof and floor in most parts remain thin and membrane-like. Quite at its anterior extremity this series of vesicles has begun to expand into that *magnum opus* of ontogenesis and organogenesis, the cerebral hemispheres: there is as yet little to be seen of that crowning superstructure of developmental activity; but its foundations are there, firmly laid, and even beginning to show above the surface. In the spinal cord, also, histological and structural differentiation is going on: spongioblasts are forming that neuroglia-network, in the meshes of which neuroblasts, potential neurons with potential axons and dendrites, begin to appear; and, having appeared, begin to stretch forth in bewildering fashion, and with far-reaching results so far as the later structure of this part of the nervous system is concerned.

Hardly less remarkable are the developments going forth in the great body cavity of the embryo, where we find an intestine with an end-gut or *pars ultima* and an entodermal cloaca (*pars penultima*), the *anlage* of a liver and pancreas, the indication of a trachea and lungs, and relatively large and well-formed Wolffian bodies. The last-named organs have, indeed, already reached their acme of structural differentiation and the culminating point of their developmental activity. The heart, also, is rapidly approaching the high degree of constructive detail which it maintains during the remainder of antenatal life; for, unlike the Wolffian bodies, it does not then start upon a degenerative progress, but remains permanently efficient and physiologically of great importance. Very soon its chambers, right and left, will be divided off from each other by septa, complete but for the presence of the foramen ovale, which serves as a communication between the auricles until the occurrence of birth and the replacement of the fœtal with the adult type of circulation.

Extra-embryonic Parts in the Fifth Week

In the fifth week after impregnation, the decidual membranes bear much the same relations to each other and to the chorionic vesicle as in the fourth week. Minot (*Human Embryology*, p. 13, 1892) describes the decidua serotina in a specimen of about this age (fifth week) as consisting of a compact and a spongy layer, the former constituting one quarter of the whole thickness of the serotina. The surface was without any trace of epithelium, but was covered with a thin fibrous and granular coagulum. The compact layer consisted almost exclusively of young large decidual cells and a clear homogeneous matrix; the cells were irregular in shape, but more or less rounded, with processes which occasionally united two cells together. In the deeper or spongy (cavernous) layer the decidual cells were not so large; they were more fusiform in shape, and had longer, less round nuclei. The spaces of the cavernous layer were glandular, and in many of them the shed epithelium (often degenerated) could be seen lying. In the smaller spaces the epithelium (columnar in type) was well preserved and still attached to the walls. The blood-vessels lay between the glands, and Minot found nothing corresponding to the "colossal capillaries" of Turner.

The chorionic vesicle is still covered all over with villi, but these are most marked in the region corresponding to the decidua serotina. The minute anatomy of the parts is the same as in the fourth week. The space between the amniotic sac and the inner aspect of the chorionic vesicle—the extra-embryonic cœlom—is still large, but not so large as in the preceding week; at the end of the fifth week it may be stated roughly that the space between the embryo and the chorion is divided equally between the amniotic cavity and the cœlom (Mall, *Journ. Morphol.*, xii. 375, 1897). The amniotic membrane is still not far removed from the body of the embryo, and possesses the same histological appearances as formerly. The umbilical vesicle is pear-shaped; its stalk has lengthened considerably, and for the first part of its course is enclosed within the Bauchstiel; and the vesicle is no longer increasing in size.

The Embryo in the Fifth Week

Various human embryos of the age of the fifth week have been described, several by His (Pr, M, Br, Rg, Ko, Sch, etc.), some by Minot and

Coste, and others by Rabl, Phisalix, R. Dorello, and Hochstetter. In length the embryo now measures from 8 to 14 mm.; probably, then, 1 cm. may be taken as representing its average length about the middle of the fifth week. The distinct C shape of the fourth week is beginning to be partly lost: the body of the embryo shows some straightening, and so the head seems as if it formed a right angle with it. Between the region of the hind-brain and the back opposite the origin of the anterior limb-bud, there is a slight concavity, called *Nackengrube* by His; this persists till the eighth week (*vide* Neo-fœtal Period, p. 109). Towards the end of the fifth week the head, as seen from the side, has an area about equal to that of the rest of the body; the cephalic and cervical flexures or bends are well marked, the latter reaching its maximum at this date. Both the Bauchstiel and the stalk of the umbilical vesicle have lengthened; they are combined in part of their extent to form the single structure, which may now be called the umbilical cord: towards the end of the week the cord shows one or two spiral turns, and contains one or more coils of intestine. The stalk of the umbilical vesicle is longer than the cord, and is disposed between the chorion and the amnion.

The limb-buds show a marked development during this period of seven days: at first they are unsegmented; then they exhibit two segments; then the tripartite division; and finally the digits are roughly indicated in outline, being still webbed. The upper limbs are generally more advanced in formation than the lower ones; for instance, they show digitation earlier. Along with their differentiation into three parts or segments, the limbs have undergone increase in size, and in embryos of about thirty-five days they project beyond the outline of the body in profile views. When the limbs are in the two-segment stage, the distal part is flattened and the proximal cylindrical, and there is a constriction between; later, a second furrow or constriction appears in the cylindrical part, dividing it into two parts. So the foot, leg, and thigh, and the hand, forearm, and arm are marked out.

The tail of the embryo is now very marked; in fact, in this (the fifth) week it reaches its maximum development, and is evidently a true tail (Fig. 17). At the other end of the embryo the cervical or præcervical sinus has greatly deepened, so that the fourth, third, and even the second branchial grooves have disappeared from the surface and lie within it. The first branchial groove is still seen; in fact it never enters into the cervical sinus, but persists externally as the external auditory meatus. Around it the six tubercles, which unite later to form the pinna or external ear, are quite recognisable. In the adjacent facial region marked changes can also be seen. The nasal pits are in process of demarcation by the growth of processes in the following manner: from the naso-frontal process its two lateral edges, the two globular processes as they are called, become thick and rounded, and form the inner boundaries of the nasal pits; and from the sides of the base of the naso-frontal process the two lateral nasal or frontal processes grow downwards and outwards, and bound the nasal pits externally, shutting them off from the depressions for the eyes. The nasal pits, however, are still in communication below with the primitive oral cavity. The lateral nasal process on each side is at first separated from the maxillary process of the same side by a groove (the naso-optic furrow or lachrymal groove), but later it unites with it and grows inward towards the middle line below the nasal pit; in the sixth week, as we shall see, it joins the naso-frontal process, and so separates the nasal pit from the oral cavity. Towards the end of the fifth week the mandibular processes unite in the middle line. About this time the optic vesicle comes into contact with the lens vesicle and is invaginated by it, becoming the secondary optic cup. The under surface of the vesicle is also invaginated, and so is that of its tubular stalk, so that an aperture (choroidal fissure) and a trough are thus formed. Mesodermic tissue grows into the optic cup through the choroidal fissure, and it also grows into the trough of the stalk (future optic nerve); in these places it gives origin later to the vitreous body and the central artery of the retina.

The only other external feature of note at this stage of development is the forward projection of the abdomen, due to the large size which the liver has now attained. It may be added that the ectoderm (or epiblast) covering the body consists of two layers: an outer one, the epitrichium, made up of slightly flattened cells which later become dome-shaped, and an inner layer of larger cells which give rise to the epidermis.

The internal arrangements may now be described, the same order of description being followed as was adopted for the fourth week.

The spinal cord, in the fifth week, is divisible into three layers: an outer neuroglia layer or marginal velum, a middle or mantle layer, and an inner or ependymal layer. These have increased in thickness, and the cells of the ependyma have become ciliated. The sympathetic system shows a further advance in formation. Two ganglionic masses are found near the omphalo-mesenteric artery, one above and the other below the vessel; these represent the ganglia of the solar plexus, and the upper ganglion is joined to the ganglionated cord by fibres representing the greater and lesser splanchnic nerves. Near the origin of the umbilical arteries is another enlargement of the ganglionated cord, which represents the pelvic plexus (inferior mesenteric and hypogastric ganglia). Cells, also, are found in the neigh-

bourhood of the stomach, and these are connected with a plexus formed from fibres from the vagus nerves. In the cervical region a plexus is being formed, composed of ganglia of cells which have wandered from the cell column and of fibres closely related with the descending branches of the vagus.

In the brain, changes are manifest. A longitudinal ridge-like thickening appears in the roof or dorsal wall of the *mid-brain vesicle* or mesencephalon; later, in the ninth week, this is replaced by a furrow, at the sides of which are developed the corpora quadrigemina, but as yet there is no trace of these bodies. In the *hind-brain* or rhombencephalon, further developments are visible now both in the floor and in the roof. The roof-plate is wide and thin; over the posterior half of the fourth ventricle it forms the inferior medullary velum, while over the anterior half the superior medullary velum develops as well as the rudiment of the cerebellum. Out of the alar and basal laminæ are formed the rudiments of the pons and medulla; the attachments of the roof-plate or velum to the alar luminæ is marked by the obex and ligula, which are thickenings on the margins of the lower angle of the fourth ventricle. The rudiment of the restiform body is found in the upper margin of the alar lamina. If we now look at the parts in front of the mesencephalon, we find that the roof-plate of the *diencephalon* or inter-brain is thin-walled and somewhat folded, while in the floor-plate is a well-marked ventral groove; the lateral walls are divided into dorsal and ventral zones by the grooves known as the sulci of Monro, which extend forward towards the point of origin of the optic evaginations. At the posterior end of the folded roof-plate is an elevation which later becomes a hollow evagination, the pineal process, and still later the pineal body

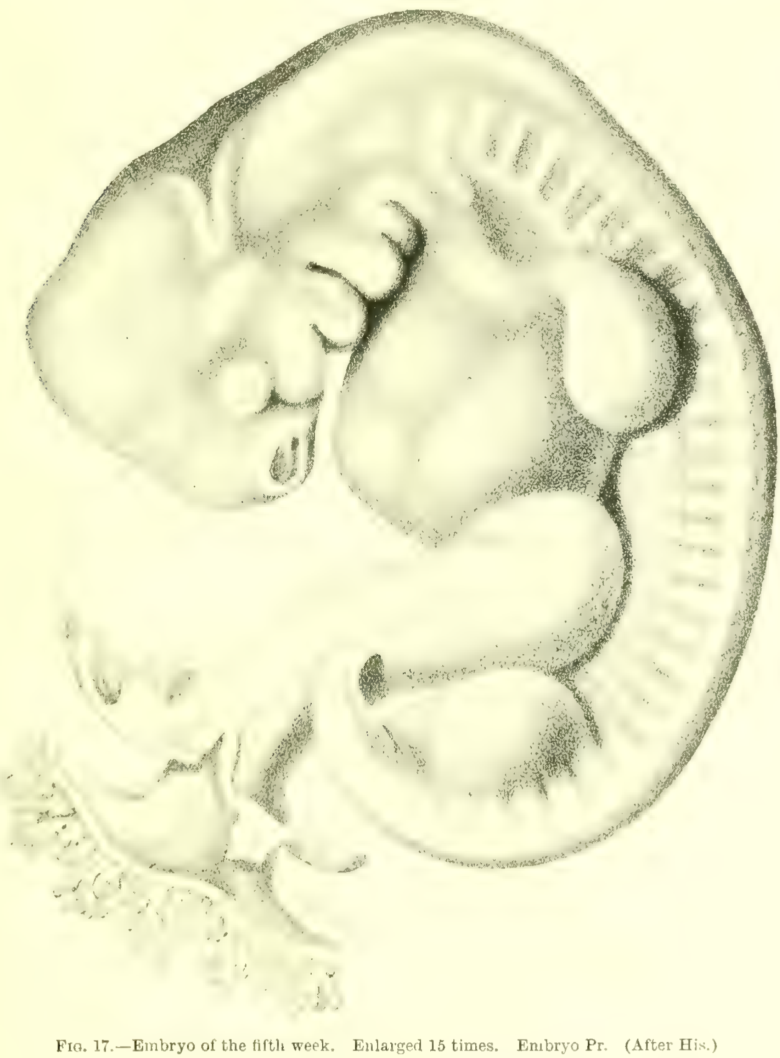


FIG. 17.—Embryo of the fifth week. Enlarged 15 times. Embryo Pr. (After His.)

or epiphysis. The rest of the roof-plate in front of the pineal elevation represents the velum interpositum of adult anatomy; it forms a ridge at first, but soon becomes a thin membrane upon which at a later date blood-vessels arrange themselves, and the whole structure is invaginated into the third ventricle as the choroid plexus. The dorsal zones of the lateral walls thicken, and towards the end of the fifth week begin to project inwards into the cavity of the ventricle as the optic thalami. Behind the optic thalami are other thickenings giving rise to the pulvinares and the external geniculate bodies. The ventral zones become the subthalamic regions, and in them or perhaps in the floor-plate are developed the corpora albicantia (mammillary tubercles). In front of the diencephalon is the *telencephalon* or hemisphere-brain. It has, as we have already seen in the end of the fourth week, a median portion which contains the anterior part of the third ventricle, and two lateral outgrowths, the cerebral hemispheres. The roof of the median portion is converted into the anterior part of the velum interpositum. From its dorsal zones arise the optic evaginations, as has already been pointed out; while the ventral zones form the anterior part of the subthalamic regions. In the floor-plate is the evagination of the hypophysis which during this week comes into contact with the invagination of the roof of the oral cavity to form with it the pituitary body. The cerebral hemispheres arise from the sides of the dorsal zones, and also get a prolongation of the roof-plate or velum interpositum. In the ventral part of the wall of each a thickening is to be noted, the future corpus striatum, and in the more dorsal part is the mantle or pallial portion of the hemisphere. The cavities of the hemispheres are, of course, the lateral ventricles, and into them project the choroidal folds which do not at this date possess blood-vessels. In this relation are to be observed the arcuate and choroidal fissures. In the floor of the telencephalon the tuber cinereum appears. The first indication of the falx cerebri is found lying in the longitudinal fissure between the two cerebral hemispheres. There are as yet no traces of the corpus callosum or fornix. The olfactory lobes can now be recognised as areas on the ventral surface of the cerebral hemispheres; they are at first hollow and later become solid, and finally (third month) give rise to the olfactory bulbs and stalks and to the olfactory tracts, trigone, and anterior perforated spaces.

The origins of the cranial nerves as they exist in the fourth week have been already sketched; but some further developments may here be noted. During the fifth week the olfactory nerves are much more distinctly indicated than in the preceding week; the olfactory ganglion is produced, its cells assume the bipolar form, and by the elongation of their poles on the one side they come into relation with the brain, while on the other side they join the olfactory epithelium. In His' embryo Ko (a fifth week specimen), the ganglia and trunks of the third (oculo-motor), fourth (trochlear), fifth (trigeminus), seventh (facial), eighth (acoustic), ninth (glosso-pharyngeal), tenth (vagus), eleventh (spinal-accessory), and twelfth (hypoglossal) nerves can all be much more clearly traced than in fourth week embryos. The sixth (abducens) nerve is composed entirely of fibres arising in the ventral or basal zone of the hind-brain; it has no ganglion. The eighth nerve has a vestibular and a cochlear branch; at this time the ear has advanced in development, the semicircular canals have commenced to form, and the twisting of the cochlea has begun. The eleventh nerve and its branches have practically their adult relations in this week of embryonic life, and the twelfth (hypoglossal) nerve passes round the sinus cervicalis and then curves forward into the tongue.

The alimentary system, in the fifth week, shows some increase in complexity. The oral cavity is not yet shut off from the nasal. The front of the tongue is marked off from the back by two oblique lines which begin at the foramen cæcum and form an open V; the anterior part is developed from the tuberculum impar, and the posterior from the region of the second and third branchial arches (ventral ends). At the opening (the future foramen cæcum) is the orifice of the duct of the median part of the thyroid gland (thyroglossal duct); it loses its lumen and begins to atrophy at the fifth week. Towards the end of the week a longitudinal ridge appears on each side in the floor of the mouth in the part known as the alveolo-lingual groove; this is the *anlage* of the submaxillary gland and Wharton's duct. The pharynx is still relatively large, being equal to about half the length of the alimentary canal. The pharyngeal pouches are beginning to undergo the series of changes which transform them into permanent structures; for example, the first pouch, on each side, is being shut off to form the tympanic cavity at one end and the future Eustachian tube at the other, the "closing membrane" persisting as the tympanum; the median lobe of the thyroid gland is related to the anterior ends of the second pair of pouches; and from each of the two fourth pouches arises a ventral prolongation or diverticulum, which becomes the *anlage* of one of the two lateral lobes of the thyroid gland. At a later date (sixth week) evaginations from the third pair of pockets give origin to the epithelial parts of the thymus gland.

The opening of the trachea into the pharynx is slit-like; in front is the *anlage* of the epiglottis, and at the sides are the arytenoid ridges whose thickened epithelium almost closes the opening; in the ridges, on each side, are two projections, the cuneiform and cornicular tubercles. There are as yet no traces of the cartilages of the larynx; these appear at the seventh week,

and the cartilages of the trachea a week or two later. Through an increase in the number and size of their lobes the lungs become much more than simple buds. A pulmonary artery can be traced to each of them.

To return, now, to the description of the alimentary system, it may be observed that during the fifth week the noteworthy flexure of the intestine below the level of the stomach is established. In this way is formed a U-shaped loop, with its closed end projecting towards the ventral body-wall; the dorsal mesentery at this point lengthens in order to permit of this formation. The vitelline duct or yolk-stalk is attached to the apex of the U-shaped loop, and may remain permanently as Meckel's diverticulum; the vitelline artery (superior mesenteric) supplies the loop. At a point on the lower border of the loop the intestine suddenly becomes larger; this indicates the caput cæcum or commencement of the large intestine. All the bowel above this point forms the small intestine, and thus it comes about that Meckel's diverticulum, when present, is attached to the ileum above the level of the ileo-cæcal valve. At first the U-shaped loop is extra-abdominal in position, being situated in a cavity formed by the somatopleure at the umbilicus; as a rule, it passes within the abdomen by the end of the second month. The angle between the U-shaped loop and the hind-gut becomes the splenic flexure, although as yet, of course, there is no trace of the *anlage* of the spleen. The hind-gut is supplied with a dorsal mesentery by which it is suspended. The inner surface of the intestine is as yet quite smooth, folds not making their appearance till the next week. It has been stated that towards the end of the fifth week the mucous membrane of the duodenum increases so much in thickness that the lumen is narrowed and finally obliterated, to reappear again at the beginning of the sixth week (Tandler). The pancreas lies between the layers of the dorsal meso-gastrium at this age, and is parallel to the greater curvature of the stomach. The part of the dorsal meso-gastrium which lies between the pancreas and the stomach becomes at a later date much elongated, to form the great omentum. The liver develops rapidly during this week, increasing greatly in size, and causing the anterior abdominal wall to project, as may be well seen in Mall's reconstructed embryo No. ix. (*Johns Hopkins Hosp. Bull.*, ix. p. 199, 1898). The gall-bladder is quite recognisable in this embryo. During this week (the fifth) the end-gut (*pars ultima* of Berry Hart) is in course of disappearance. The entodermal cloaca (*pars penultima*) is now divided into an interior and a posterior division, the former forming the bladder and urinogenital sinus, and the latter the rectum. In the sixth week both the bowel and urinogenital sinus communicate with a large space, the cloaca, best known as the ectodermal cloaca. It may be conjectured, for we do not know with certainty, that this cloaca has been formed during the fifth week by the breaking down of an ectodermic pyramidal plug with its apex towards the lower part of the urinogenital sinus meeting the coronal partition of the primitive gut (Berry Hart, *loc. cit.*). Apparently the communication of the cloaca with the exterior thus established becomes shut off again between the seventh and eighth weeks by Reichel's anal tuberosities (*Analhöcker*).

The urinogenital organs have advanced considerably in their ontogeny since the fourth week. The mesonephros or Wolffian body is still well developed and large. It extends as a ridge on each side of the spine from the posterior cervical region to the pelvis. To its inner side lies the genital ridge, and it is possible, according to Nagel (*Arch. f. mikrosk. Anat.*, xxxiv. 269, 1889), even at this age (fifth week) to determine by microscopical examination whether the genital gland is male or female; if it is to be an ovary the large germinal cells (primitive ova) are numerous, and the germinal or cœlomic epithelium forms a thick layer of several rows of cells, while if it is to be a testicle the large germinal cells are fewer, and the cells of the epithelium are arranged in groups rather than in a distinct band or layer. The convoluted tubules and glomeruli of the Wolffian bodies open into the Wolffian ducts, and these in their turn open into the entodermal cloaca. As has been already pointed out, the ureter is present at the end of the fourth week as a small bud attached to the Wolffian duct just above its opening into the cloaca; in the fifth week this bud develops a stalk and becomes the permanent kidney and duct (ureter) which lie in the lumbar region behind both the Wolffian body and the peritoneum. The permanent kidney or metanephros, then, is developed from this outgrowth from the Wolffian duct; at the same time it is believed that it obtains part of its structure from a condensation of the mesenchyma which surrounds it, to which the name of metanephric blastema has been given. Possibly the blastema only contributes the connective tissue and vessels of the kidney. About the end of the fifth week, or perhaps in the sixth week (the time is not known with certainty), the Müllerian ducts appear as peritoneal evaginations in the ridge or folds that lie on the ventral surface of the Wolffian bodies. The Müllerian ducts come to open into the urinogenital canal posteriorly, while anteriorly they have a free communication with the peritoneal cavity. A trace of the external genitals is visible during the fifth week in the shape of the genital tubercle, a small eminence on the ventral wall of the urinogenital sinus. At this time, also, the suprarenal capsules or bodies are quite recognisable, and each is found near the cephalic end of the mesonephros or Wolffian body of the same side. Their relation to the kidney is a

later acquired one. They are probably derived from the Wolffian bodies; but a part (medulla) has origin in the sympathetic nervous system.

The heart and circulatory system are now well advanced in development. The auricles and ventricles are not yet fully separated into right and left chambers. The opening of the sinus venosus into the right auricle is guarded by right and left lateral valves, which prevent regurgitation of blood when the auricle contracts. At a later date the right valve becomes the Eustachian and Thebesian valves; probably the left forms part of the auricular septum (primary). Even as early as the fifth week a trace of the secondary septum (or septum spurium) of the auricles can be made out lying to the right of the primary one. The auriculo-ventricular canal is divided into right and left auriculo-ventricular orifices by two structures known as the anterior and posterior endocardial cushions. The bulbus arteriosus is divided into an anterior or pulmonary and a posterior or aortic part by anterior and posterior ridges (anterior and posterior aortic septa), which fuse together while their lower ends unite with the interventricular septum. The resulting aortic part retains its connection with the fourth left aortic arch, which becomes the ascending part of the arch of the aorta; the pulmonary or anterior part is continuous with the fifth arch of the left side and constitutes the ductus arteriosus (or ductus Botalli), with branches to the two lungs. It is not known how this change in the relations of the pulmonary arteries, so that both arise from the fifth left arch, is brought about (Minot). The fifth arch on the right side atrophies; the right fourth arch persists as the right subclavian and innominate arteries; the third arch on each side remains as the internal carotid. Now, therefore, the third and fourth arches and the vessels representing the first and second arches are all connected with the left side of the heart through the aorta, while the left fifth arch is brought into relation with the right ventricle through the pulmonary trunk. The segmental arteries and the vessels for the limbs doubtless undergo important modifications during this week, but these are not yet clearly known. The arrangement of the veins is probably similar to that described for the fourth week; the origin of the vena cava inferior has not yet been clearly ascertained for the human embryo. The blood at this age contains only nucleated red blood corpuscles or erythrocytes.

The sustentacular parts of the embryo show further developments at the fifth week. For instance, in the limbs the fibrous basis of some of the future bones can be recognised, and here and there chondrification has begun. In the pelvis, also, the three parts of the *anlage* of the os innominatum (ilium, ischium, pubes) are present; so are the membranous ribs and clavicle, etc. According to Arthur Thomson (Cunningham's *Text Book of Anatomy*, p. 180, 1902), ossification may be seen in the clavicle as early as the fifth week. Meckel's cartilage is present in the mandibular arch.

It must be admitted that there is much that is uncertain about human ontogenesis in the fifth week after impregnation. In this period take place several complicated rearrangements of parts; and just how these rearrangements are effected we do not clearly understand. This remark applies specially to the changes which occur in the branchial arches and clefts, in the pharyngeal pouches, in the great veins of the abdomen and liver, and in the pleuro-peritoneal relations. We do not know how these changes take place, neither do we know with any degree of certainty when they occur. Consequently any attempt to place before the reader a chronological account of the embryology of this period has to face the double difficulty of uncertainty as to the facts and ignorance regarding the dates. There is also the constant problem of the recognition of parts; for when one, so to say, loses sight of a structure in the embryo for a day or two it is not easy to recognise it again at a later stage in a new form and with new relations. The whole problem, therefore, becomes a very complex, involved, intricate, and perplexing one; yet, in order to preserve unbroken the chronological account of human embyronic life, I felt myself constrained to put before my readers such facts as I could gather together from reliable authorities. They are few in number and obscure, but they must serve till further light has been thrown upon this feebly illumined part of ontogenesis. It may be said, in parenthesis, that the nomenclature of embryology does nothing to render this obscure portion of the subject less dark; in fact, the giving of two, three, or even four different names to the same structure is irritatingly confusing.

Although there is much that is obscure about organogenesis in the fifth week, there is also not a little that is well known. The embryo begins to lose something of the exaggerated C shape that it had in the preceding week, and attains to the length of 1 cm., being now about one-fiftieth of the length of the mature fœtus. Much has yet to be accomplished before the 1 cm. embryo is converted into the 51 cm. fœtus, but it may be doubted whether any of the superadded centimetres are accompanied by such a marvellous exhibition of developmental activity as is this first one. Among the many phenomena of the week we must note the assumption by the Bauchstiel and yolk-stalk of a truly cord-like character, the transformation of the bud-like extremities into evident and segmented limbs, and the presence of a decided and indisputable tail. The face is now more face-like, having a lower jaw united in the

middle line, nasal pits which, although incomplete, suggest a nose, and recognisable eyes, but with no eyelids or even *anlages* of eyelids. The embryo is beginning to be dimly human in appearance, a change which is aided by the disappearance of the second, third, and fourth branchial arches and grooves into the sinus præcervicalis whose margins close over and entomb them.

In its internal arrangements the embryo is rapidly passing towards a more mature distribution of structures and organs. The origins of cranial nerves can be recognised; the pituitary body is present, so are suprarenal capsules and a sympathetic system with a solar plexus; the bowel has thrown itself into a U-shaped loop, and given thus an indication of its coming division into small and large intestine; the thyroglossal duct is beginning to close; the ureters are present as little buds from the Wolffian ducts; and in the genital ridges histological evidence can be obtained of the sex to which the embryo belongs. In the region of the brain, also, the *anlages* of numbers of new parts and structures have appeared, including those of the medullary velum, pons, velum interpositum, optic thalami, corpora striata, tuber cinereum, and falx cerebri. Finally, the surface of the whole body is covered by the epitrichium, that mysterious layer whose fate and whose functions are alike unknown.

The Sixth Week

The sixth week after impregnation is the last of typically embryonic life. It is not that there is no further organogenesis after this period, as a matter of fact some organogenesis is going on up to the full term of antenatal existence and even after it; but the sixth week marks the close of the epoch of almost purely constructive processes. By this time all the great formative procedures have been initiated; the plan of the human embryo has been, as it were, fully indicated; some complicated rearrangements have also been effected; and the embryo may now be called the transition-organism, for although it is not yet a fœtus it is no longer simply an embryo. We may call it the neo-fœtus. There is still much to be done: many of the organogenetic processes have to be carried to completion; a great deal of histological differentiation has to be effected; many parts have yet to assume their mature relations with each other and with the framework of the body; the organism has to grow from an embryo measuring little more than half an inch in length to a fœtus of twenty inches; and modifications have to occur in the manner in which its organs carry on their functions, and in the relations which they bear to the maternal economy. Nevertheless, for the organism as a whole, truly embryonic life has nearly finished; and what remains to be done is no longer development but growth and histological and physiological differentiation.

Extra-embryonic Parts in the Sixth Week

J. C. Webster gives a good and detailed account of the extra-embryonic parts of the uterine contents at the sixth week (*Human Placentation*, pp. 15, 28, 38, etc., 1901); upon this the following description is based.

The decidua vera varies from 3 to 7 mm. in thickness, and can be readily divided into a compact and a spongy layer. The spongy part can be subdivided into an outer part with slightly branched glands, and a deeper portion where there is much branching. The lining epithelium is still to a great extent present, although somewhat altered: the columnar cells are now cubical or flattened; their nuclei are rounded or flattened in shape, and some have degenerated; and the cilia have disappeared. The glands have increased in size, but there is no complete evidence of any new formation of them; their mouths (in the compact layer) are narrowed or obliterated; their deeper parts are much enlarged; their epithelium is generally cubical or flattened, very rarely columnar, and in many instances is shed in masses or in individual cells. In the interglandular tissue the most marked change is the development of the decidual cells: this change is practically limited at present to the compact layer, and is most evident in its outer portion; the decidual cells are rounded, oval, polygonal, or spindle-shaped, with large and somewhat rounded nuclei, and in most places they are connected by broad or slender processes; and here and there they are seen with two nuclei, probably indicating the occurrence of cell-division. In the compact layer the capillaries in some parts are enormously dilated, in other parts there is only a moderate increase in size; here and there are small extravasations of blood in the decidual tissue.

The decidua reflexa (capsularis) has not yet come into contact with the vera, so that a uterine cavity still exists, although it is diminished in size by the growth of the embryo and its envelopes. The basal and polar part of the reflexa can be distinguished: the former consists of an inner compact and an outer spongy layer which contains gland spaces, not so numerous as in the corresponding layer of the vera or serotina; the latter or polar portion contains very few or no glands. None of the glands open on the inner surface of the reflexa. Blood spaces are to be seen throughout the membrane; in the basal part some communicate with the intervillous spaces of the opposed chorion læve; and here and there masses of syncytium may be traced into the vessels. At this age there are already signs of degeneration in the reflexa, of the nature of coagulation-necrosis of the decidual tissue, so that in the innermost part is found a layer of fibrin-like

material thickest at the pole. Possibly some of the fibrin may arise from the blood. On the inner surface of the reflexa no trace of maternal epithelial cells can be seen, and on the outer surface is an epithelium with much the same characters as that on the vera (cubical or flattened, not ciliated).

The decidua serotina is from 2 to 3 mm. in thickness, and the compact layer measures about one-quarter of the whole. In some parts the whole decidua looks as if made up of the compact layer, but as Webster points out this may be a variation of no importance. The surface is irregular and no surface epithelium is visible. The outer parts of the glands in the compact layer are obliterated, and both in them and in the deeper parts in the spongy layer the lining epithelium may be cubical or irregular, attached or loose, and more or less degenerated. In the interglandular tissue characteristic decidual cells are found in some parts, and in others they show signs of degeneration. The superficial part of the compacta shows an irregular layer of fibrin-like material; this has to be distinguished from the layer of fibrin on the surface which results from coagulation of the blood. At intervals chorionic villi are fixed to the surface, and between them flattened masses of protoplasm (syncytium or plasmodium) are attached. The syncytium forms a more broken layer than it did before. There are no cell outlines, and the nuclei are arranged either in rows or quite irregularly. Here and there the masses of syncytium are thickened and resemble very large multinucleated giant cells, with prolongations running into the intervillous spaces. It is to be noted that no structure at all like this is to be found on the surface of the decidua vera, one among several reasons which lead us to believe that it is of fœtal origin. Processes of it extend into the substance of the decidua as deep as the muscular coat. In the spongy layer of the serotina are numerous small tortuous arteries; in the compact layer the arteries cannot be distinguished from the veins; there are numerous sinuses which may be dilated capillaries, and some of them can be seen to open into intervillous spaces and to contain masses of syncytium attached to the wall.

The surface of the chorion which is opposed to the decidua serotina is thickly covered with much branched villi. They show a thick layer of syncytium and a comparatively thin underlying cellular layer. Both on the general chorionic surface and on the villi are little projections of the syncytium. The mesoblast of the chorion and that which forms the cores of the villi has a denser and a more fibrillated appearance than in the earlier weeks, and here and there it forms a sort of basement membrane under the cellular or Langhans' layer. In the smaller villi the mesoblast is of a mucoid type; and capillaries exist in most of them, whether small or large. Among the villi are irregularly shaped pieces of syncytium, and clumps of decidual cells with pieces of Langhans' layer attached to them. The decidual masses may show signs of fibrinous or hyaline degeneration. The attachment of the villi to the serotina seems to be by means of the proliferated mass of cells of Langhans' layer at the tips; apparently the syncytium has nothing to do with it. The villi are attached at no regular intervals, by a main stem or by branches, and at right angles or obliquely to the surface. From the chorionic surface which is opposed to the decidua reflexa not nearly so many villi spring; they are less branched, show very few bud-like projections, are poorly supplied with capillaries, and sometimes contain connective tissue which is swollen or hyaline.

From the foregoing description it will be learned that the decidual and chorionic investments of the embryo have not altered greatly from what existed in the two preceding weeks. The space, however, between the vera and reflexa is much smaller, and the reflexa already has begun to show retrogressive changes. The glands, also, of all the decidual membranes are in a more or less marked state of degeneration; this is true even of the serotina, in which, however, the other parts are as yet in full activity. There is a still further development of the chorion and its villi opposed to the decidua serotina; and, although there is not here a separate and recognisable placenta, there is the physiological equivalent of that organ. The important part played by the fœtal tissues in the construction of the placenta is also quite apparent.

The amniotic sac has now increased with the marked growth of the embryo, so that the space (extra-embryonic cœlom) between it and the inner surface of the chorionic vesicle has practically disappeared. The histological characters of the membrane scarcely alter, but the matrix between its two layers becomes somewhat condensed and fibrillated. The yolk-sac or umbilical vesicle has much the same size as in the fourth week; it is obviously being left behind by the embryo in the progress of ontogenesis, for its useful part is played already. Consequently the vitelline circulation is atrophying. The great characteristics of the extra-embryonic structures in the sixth week are the growing importance of the chorionic villi which face the decidua serotina, with the consequent establishment ere long of a true and anatomically recognisable placenta, and the dwindling of the decidua reflexa and umbilical vesicle. One of the great rearrangements of ontogenesis is in progress. By its means intra-uterine existence is to be prolonged for seven months or more.

The Embryo in the Sixth Week

The embryo at the sixth week measures from 14 to 17 or 18 mm. in length. The increase in

length during this and the preceding week is in part due to the straightening of the body and the raising of the head. The changes in the external appearances of the embryo are also largely caused by this alteration in attitude. The neck bend becomes in consequence more rectangular, and the concavity at the back of the neck (*Nackengrube*) less marked. The mid-brain comes to lie more directly above the hind-brain. The abdomen is still more prominent than it was in the fifth week, with the result that the limbs do not in profile views reach to the outline of it. Other external features at this age are: the presence of a distinct tail, which, however, is relatively smaller than before; the indication towards the end of the week of slight folds surrounding the conjunctival area, the *anlages* of the future eyelids; the appearance of the fingers as separate outgrowths; the existence of a recognisable external ear or concha; the completion of the upper part of the face by the union of the two maxillary processes; and the direction of the long axes of the limbs at right angles to that of the body. The umbilical cord is now quite distinct in its whole length, and still evidently contains a loop of intestine.

If we look more closely at the face at this period of embryonic life, we shall see that through the union of the maxillary processes with the lateral nasal and fronto-nasal processes the external boundaries of the nostrils have been marked out and the mouth cavity shut off from the nasal pits. There is consequently a recognisable but very broad nose, the nasal pits being the anterior nares. It is noteworthy, also, that through the downward growth of the fronto-nasal process the mouth receives a new upper boundary. Posterior to this process the oral and nasal cavities freely communicate, for the two lateral halves of the palate do not begin to unite till the eighth week. The oral surface of the upper jaw already (towards the end of the sixth week) shows two parallel ridges, which afterwards become the upper lip and the gum. At the posterior end of the embryo the genital tubercle is now clearly recognisable; behind it is the cloacal fossa, and encircling the fossa is a ridge (genital ridge of the external genitals); no groove has yet appeared on the under surface of the genital tubercle. The histological examination of the skin shows the same two layers as in the fifth week with an indication of the dermis or cutis beneath them.

There are no very radical changes to be noted in the internal conditions of the embryo in this week. As has been already stated, most of the embryonic rearrangements have been initiated and many of them have been almost carried through. Such changes as there are affect specially the brain and spinal cord, the ear and eye, the genito-urinary organs, and the sustentacular framework of the body.

The alterations in the spinal cord are evident. The central canal has changed from an oval running antero-posteriorly into a somewhat rhomboidal form. The lateral angles of the lozenge-shaped space thus produced divide the lateral walls very distinctly into two zones or laminæ, an alar or dorsal and a basal or ventral. The roof- and floor-plates (mid-dorsal and mid-ventral laminæ) show no changes; but the alar and basal laminæ of the lateral walls do so. The posterior nerve-roots, of which there were indications at the fourth and fifth weeks, can now be recognised passing into the alar lamina; while the anterior or ventral roots take origin in the ventral lamina. At present the cells of the ventral zone are much more numerous than those of the dorsal, so that the rudiment of the anterior horn of grey matter is much larger than that of the posterior horn. There is also some sign of the developing white columns of the cord: the grey matter of the anterior horn is separated from the ventral surface of the cord by a zone in which longitudinal nerve fibres are found, the commencing anterior white column; in a less marked fashion the posterior white columns have begun to be evident between the posterior horn and the surface. There is, as yet, little or no trace of lateral columns, and there are no signs of an anterior or a posterior fissure. About this time the two ventral arteries unite into a single median vessel (arteria sulci). The whole of the complicated arrangement of bundles of fibres in the cord comes into being later than the sixth week, that is to say in the fœtal and not in the embryonic period of ante-natal life.

The changes in the brain are of the nature of continuations of those initiated in the fifth week, and the reader may refer to the preceding chapter for the account of them. There are some slight indications of further structural elaborations to which an allusion may be made. In the hind-brain or rhombencephalon and in that part of it (myelencephalon) which becomes the medulla oblongata, there is a broadening out of both roof-plate and dorsal part of the brain cavity so that the margins of the dorsal zone or alar lamina have a lateral position and are bent towards the ventral aspect to form a reflected lip (*Rautenlippe*). In the sixth week this lip continues to grow towards the ventral aspect of the myelencephalon to which it is united, and it thus buries parts (*e.g.* tractus solitarius) which primarily lay on the surface. Through this bending, the surfaces of the dorsal zone come to be dorsal and ventral instead of internal and external; from it the future restiform body is formed. The ventral zones show marked thickening, and so the floor of the cavity of the myelencephalon (fourth ventricle) is flattened. The grey matter of the ventral zones forms the formatio reticularis; it is surrounded by great numbers of neuroblasts (future olivary

bodies, etc.). Probably somewhat similar changes occur in the metencephalon and mesencephalon. The form and divisions of the diencephalon, as described under the fifth week, are maintained; the pineal body or epiphysis, a product of the roof-plate, is now more evident; there is also an expansion of the subthalamic regions, which probably are the continuations forward of the formatio reticularis. In the telencephalon, the lamina terminalis, to which reference has already been made and which probably represents the roof-plate of this part of the brain, becomes thickened and constitutes the *anlage* of the septum lucidum, the corpus callosum, the fornix, and the anterior commissure. It is not till much later that a cavity forms in the septum lucidum, to be known as the fifth ventricle. The cerebral hemispheres are now growing rapidly and project forwards and upwards in front of the diencephalon; their communications with the third ventricle (foramina of Monro) do not enlarge, but their walls thicken, and therefore the connecting structures enlarge; a commencement of the delimitation of the hemispheres into lobes is effected by the development of the fossa of Sylvius which marks off a frontal lobe and a post-Sylvian lobe (future temporal and occipital lobes), each of which contains part of the lateral ventricle. About the sixth week the olfactory ganglion on each side unites with the bulbus olfactorius of the olfactory lobe of that side.

With regard to the eye, it may be noted that in this or in the succeeding week (the seventh) the choroidal fissure disappears by a fusion of its lips. The ocular tunics also begin to form; thus mesodermic tissue surrounds the lens-vesicle and constitutes the tunica vasculosa lentis. In this tunic, vessels are developed which are associated with the hyaloid artery of the vitreous humour. The position of the eye changes: at first it was lateral and high up; in the fifth week it descended somewhat; and, now, in the sixth week it moves round more to the front: but even at the end of the second month the axes of the two eyes are far from parallel. In the middle ear the malleus and incus and their relations with Meckel's cartilage can be recognised; the cartilage of the second branchial arch is also present with the seventh nerve close behind its posterior end, and with a ring of cartilage above it, representing the stapes. The internal ear shows a differentiation into semicircular canals, cochlea, endolymphatic duct, sacculus, and utricle. From six tubercles and a ridge lying posterior to them the external ear (pinna or concha) is taking form. One tubercle becomes the future tragus, another the antitragus, another the lobule, another the antihelix, and the two remaining ones along with the ridge become the helix.

The alimentary system, like the nervous, shows no marked new developments during this week; in fact, with the exception of the development of the rectum, anus, and perineum, and the rearrangement of the intestinal coils, this part of the body of the embryo may be said to have taken on its permanent form. In the mouth the *anlage* of the submaxillary gland is now more evident than it was in the fifth week; and traces of the *anlages* of the parotid and sublingual glands may perhaps have appeared. The dental shelf is visible as a horizontal outgrowth from the epithelial downgrowth which gives rise to the lip groove. In the third pair of pharyngeal pouches the epithelial part of the thymus gland is beginning to form. There is as yet no trace of the tonsils. The œsophagus is relatively longer than before, and the stomach shows more distinctly its greater and lesser curvatures. The duodenum is clearly marked off as the part of small intestine which passes from the pyloric end of the stomach towards the dorsal body-wall. Below this is the U-shaped loop already described with the indication of the cæcum. About this time the lower limb of the U so alters its position that it comes to lie above the upper limb, and thus the cæcum, transverse colon, and descending colon come to occupy their adult relations to the small intestines. The descent of the cæcum and the development of the ascending colon, however, have yet to take place. Five other primary coils of the small intestine, in addition to the duodenal one, can be recognised; all these five lie within the umbilical cord at present (Mall, *Johns Hopkins Hosp. Bull.*, ix. 197, 1898). The ectodermal cloaca is now present, but of this more must be said in the next paragraph.

Into the ectodermal cloaca open the urino-genital sinus and the bowel. The bowel, however, which opens into the cloaca does not apparently constitute the permanent anus. The cloaca, it would seem, is closed again, and the perineum formed by the growth of two eminences (Reichel's *Analhöcker*) at the sides of it. In the present week it communicates with the exterior, and into it opens the urino-genital sinus. The Wolffian ducts in turn open into the urino-genital sinus. The two Müllerian ducts can be recognised at this age; they lie in the genital cord along with the Wolffian ducts, and have apparently in part coalesced into one duct (Berry Hart, *Trans. Edinb. Obst. Soc.*, xxvi., 264, 1901); in the upper part of their course they are separate. The kidneys and ureters are present; the latter have as yet no connection with the bladder. The Wolffian bodies may even now show signs of regression.

Little need be said regarding the respiratory and circulatory systems in this week of embryonic life. The larynx is recognisable as the dilated part of the trachea which opens into the pharynx; and the arytenoid ridges are better marked than in the preceding week. In the

heart the interventricular septum, the endocardial cushions, and the aortic ridges meet and are united by a thin membrane, the pars membranacea septi; thus the ventricles are separated. The sustentacular system shows marked progress; if the clavicle has not yet began to ossify, it may show signs of doing so now; ossification begins also in the lower jaw around Meckel's cartilage; the ribs and basis cranii are undergoing chondrification; and cartilage is evident in the bodies of the vertebræ. According to some authorities the *anlage* of the spleen may be seen even at the fifth week as a thickening of the mesoderm (mesenchyma) on the dorsal surface of the mesogastrium; according to others it is not recognisable till the eighth week.

The sixth week is, as has been stated already, the closing period of purely embryonic life. We expect, therefore, to find in it some slackening in the rate of development, and some diminution in the variety of organogenetic processes. There are fewer surprises of evolution; and there is a greater tendency for the constructive procedures to follow in their progression the lines that have been already indicated. The period of rapid transformations and of bewildering rearrangements and alterations is past.

In the extra-embryonic parts it is evident that the typically decidual environment has reached the culminating point of its functional importance and usefulness; already there are abundant signs that in future it is to be one part only of the caducal membranes (the decidua serotina) that is to pursue the path of elaboration and structural differentiation, while the others follow retrogressive ways. The preponderating importance of the chorionic villi of the chorion frondosum is becoming more and more evident, while the dwindling of the umbilical vesicle, its attachments, and its circulation is clearly recognisable. The *placenta* looms big in the immediate future of antenatal affairs. There has also been so marked an expansion of the amniotic sac and its contents (liquor amnii and embryo) that its walls are now close to those of the chorionic vesicle, and the extra-embryonic cœlom has disappeared.

The embryo has lost a little more of its exaggerated C shape; its abdomen has become more prominent and its tail less so; it has recognisably separate fingers, although its toes are not so far advanced; the mouth and nose are shut off from each other externally, and so there is a face; the eyes are coming round more to the front; the tubercles which go to form the external ear or pinna are grouping themselves so as to shadow forth that structure; and, at the other end of the body, the genital tubercle, which is later to give origin to the penis or the clitoris, is recognisable. By an organogenetic rearrangement, the details of which are unknown to us, the entodermal cloaca has become transformed into the bladder and other parts, and an ectodermal cloaca open to the exterior has appeared.

In the embryonic interior some further differentiations are in progress. The *anlages* of the salivary glands, the thymus, the larynx, the kidneys, and perhaps the spleen have appeared; the complicated changes (possibly including ossification) which lead to the disappearance of Meckel's cartilage and the formation of the auditory ossicles have begun, and ossification has commenced in the clavicle. The intestinal coils have made their great rotation with resulting complexity of arrangement of themselves and of their mesentery. The spinal cord begins to show traces of its mature division into grey and white matter, or at least into the *anlages* of the grey horns and of the white columns; and the posterior roots of the spinal nerves are forming. In the brain the signs of the great hemispherical expansion are still more evident than in the preceding week; the fossa of Sylvius with its adumbration of innumerable fissures and convolutions yet to be evolved is to be recognised; and the pineal body and the *anlage* of the corpus callosum are present.

Organogenetic Rearrangements

Before I pass from this brief sketch of development during the embryonic period to describe very shortly the neofœtal state, it may be well if I recapitulate shortly some of the more complicated organogenetic rearrangements. The chronological method does not lend itself to the description of intricate phenomena of ontogenesis extending over prolonged periods, hence the necessity of recapitulation and grouping. The embryological changes described in the following paragraphs are not all completed in the embryonic period of antenatal life, some of them indeed are in progress late in the fœtal epoch, but they are placed in association for the sake of clearness and fulness.

The Branchial Grooves and Pharyngeal Pouches.—The structures derived from these parts are:

1. From the first, the salivary glands, the external auditory meatus, the tympanic cavity, and the Eustachian tube;
2. From the second and the neighbouring parts, the tonsils, the middle lobe of the thyroid gland, and the posterior third of the tongue;
3. From the third, the thymus gland and a parathyroid;
4. From the fourth, the lateral lobes of the thyroid gland, and another parathyroid; and
5. From the fifth, the little known post-branchial bodies.

The Branchial Arches.—The *osseous and cartilaginous structures* derived from them are:

1. From the first, the maxillary process and its derivatives (superior maxilla, zygoma of temporal, malar, internal pterygoid process of sphenoid, and palate), and the mandibular

process and its derivatives (lower jaw, and Meckel's cartilage with the malleus and incus);

2. From the second, the stapes, the styloid process, the stylohyoid ligament (which may ossify), the lesser cornu of the hyoid bone, and probably part of the body of that bone;

3. From the third arch, the greater cornu and the rest of the body of the hyoid;

4. From the fourth, probably the thyroid cartilage of the larynx; and

5. From the fifth, possibly a part of the thyroid cartilage.

The *nerves* of the arches are:

1. Of the first, the second and third divisions of the fifth;

2. Of the second, the seventh and eighth;

3. Of the third, the ninth or glosso-pharyngeal;

4. Of the fourth, the superior laryngeal branch of the tenth nerve or vagus; and

5. Of the fifth, the inferior laryngeal branch of the vagus.

The *arteries* of the branchial arches, consisting of five [1] aortic arches on each side, arising from a ventral stem and joining a dorsal one, are transformed into the following permanent arteries:—

1. The first aortic arch disappears, unless the internal maxillary artery represents it, and the ventral stem becomes part of the external carotid, while the dorsal stem becomes part of the internal carotid;

2. The second aortic arch also degenerates, to be indicated possibly by the future lingual artery;

3. The third arch along with its dorsal stem and that of the second and first arches becomes the internal carotid artery;

4. The fourth arch on the right side becomes the first and second parts of the subclavian artery and its lower part disappears, while on the left side it forms the permanent aortic arch;

5. The fifth arch on the right side in great part disappears, while that of the left side remains as the ductus arteriosus.

The Cerebral Vesicles.—Each of the five cerebral vesicles consists primarily of five parts, —a roof-plate, a floor-plate, an alar lamina or dorsal zone of the lateral wall on each side, a basal lamina or ventral zone of the lateral wall on each side, and a cavity. Each of these five parts may have a representative or representatives in later development.

1. *The Myelencephalon.*—From the myelencephalon, which, as a whole, becomes the medulla oblongata, the following are derived:—

(*a*) From the roof-plate, the posterior velum.

(*b*) From the floor-plate, the median raphe.

(*c*) From the alar laminæ, the nuclei of the sensory roots of some of the cranial nerves, the nuclei of Goll and Burdach, and the olivary bodies.

(*d*) From the basal laminæ, the nuclei of the motor roots of some of the cranial nerves, and the formatio reticularis.

(*e*) From the cavity, the posterior part of the fourth ventricle.

[1] Some embryologists maintain that there are six.

2. *The Metencephalon*, as a whole, becomes the cerebellum and pons. Its connection with the mesencephalon constitutes the isthmus rhombencephali. Both these parts are represented by certain structures; therefore those arising from the isthmus are placed separately—

(*a*) From the roof-plate, part of the posterior velum and the vermis of the cerebellum.

(*b*) From the floor-plate, the median raphe (?).

(*c*) From the alar laminæ, the lobes of the cerebellum, the flocculi, the nuclei of the sensory roots of some of the cranial nerves, and the nuclei of the pons (?).

(*d*) From the basal laminæ, the nuclei of the motor roots of some of the cranial nerves and the formatio reticularis (?).

(*e*) From the cavity, part of the fourth ventricle.

2*a*. *The Isthmus Rhombencephali* gives origin, in all probability, to the following structures:—

(*a*) From the roof-plate, the anterior velum and the valve of Vieussens.

(*b*) From the floor-plate, the median raphe.

(*c*) From the alar luminæ, the superior peduncles of the cerebellum or brachia conjunctiva.

(*d*) From the basal laminæ, the posterior part of the crura cerebri and the posterior part of the tegmentum (?).

(*e*) From the cavity, part of the fourth ventricle.

3. *The Mesencephalon*, as a whole, gives rise to part of the crura cerebri and to the corpora quadrigemina, but it is not exactly known in what manner. The following is an incomplete and tentative arrangement:—

(*a*) From the roof-plate, some part not identified.

(*b*) From the floor-plate, the median raphe.

(*c*) From the alar laminæ, the corpora quadrigemina.

(*d*) From the basal laminæ, the nuclei of the third and fourth cranial nerves, the anterior part of the tegmentum, and the anterior part of the crura cerebri.

(*e*) From the cavity, the iter or aqueduct of Sylvius.

4. *The Diencephalon* or *Thalamencephalon*, which, as a whole, constitutes the optic thalami, the corpora albicantia, and the pineal body, gives rise in detail to the following parts:—

(*a*) From the roof-plate, the velum interpositum and the pineal gland or epiphysis.

(*b*) From the floor-plate, the tissue of the mid-ventral line.
(*c*) From the alar laminæ, the optic thalami, the pulvinares, and the external geniculate bodies.
(*d*) From the basal laminæ, the subthalamic region, the corpora albicantia (corpora mammillaria), and the tuber cinereum (part).
(*e*) From the cavity, part of the third ventricle.

5. *The Telencephalon*, which, as a whole, gives rise to the cerebral hemispheres, corpora striata, olfactory lobes, and infundibulum, is converted into the following detailed structures:—
(*a*) From the roof-plate, the anterior part of the velum interpositum (in the median part of the telencephalon) and the floor of the choroidal fissure (in the hemisphere part).
(*b*) From the floor-plate, the infundibulum and part of the pituitary body (hypophysis).
(*c*) From the alar laminæ, the lamina terminalis and optic evaginations (in the median part), and the pallium or mantle, the corpora striata, and the olfactory lobes (in the hemisphere part).
(*d*) From the basal laminæ, the anterior part of the subthalamic region and of the tuber cinereum.
(*e*) From the cavity, the anterior part of the third ventricle, the lateral ventricles, and the foramina of Monro.

The Neofœtal Period

Just as postnatal life begins with a period of transition or readjustment to suit new environmental conditions, a period named the neonatal; so the passage from embryonic to fœtal life is marked by a transition time of adaptation (*Natura non facit saltus*—Nature makes no leaps) which we may call the neofœtal, during which, among other notable phenomena, the placental economy is being established. The neofœtal period coincides roughly (there are no sharp limits, Nature, as has been said, making no leaps) with the second half of second (lunar) month of intra-uterine life. Its commencement is on or about the fortieth day (end of sixth week), when the new organism takes on a form which can be recognised as distinctly human; this somewhat indefinite change Minot regards as marking the end of the embryonic epoch (*Human Embryology*, p. 391, 1892) and the beginning of the fœtal. It is, however, better to regard it as marking the beginning of a period which is neither embryonic nor fœtal, but a transition between or combination of the two—the organism is putting off its distinctively embryonic and putting on its fœtal characters, is becoming *human*, *i.e.* recognisably similar to child or adult. The "transition" form is seen in His' embryo xxxiv. (Dr), the estimated age of which was thirty-eight days, and the length of which from neck-bend to coccygeal bend was 1·5 cm. (Fig. 18). "Transition organism," we may call it, yet it is probably more correct to regard organisms of all ages between six weeks and two months as transition forms, the transition itself being not sudden, but gradual,


Fig. 18.


Fig. 19.

requiring two weeks at least. For, during the seventh and eighth weeks (neofœtal period), several changes take place in the appearance of the organism; and some of these can be recognised by comparing the His embryo (Fig. 18) with a fœtus (Fig. 19) in my collection, measuring 2·5 cms. in length (cephalo-coccygeal length), and of an estimated age of fifty-six days (end of neofœtal period).

Anatomy and Physiology of Neofœtal Period

The changes which occur in the neofœtal period are external and internal; they are less marked than those which have occurred in the embryo, but they are much more marked than those that are to occur in the fœtus.

With regard, in the first place, to *external* appearances, the following may be emphasised as noteworthy. The greater part of the head of the six weeks' embryo is sharply flexed at right angles to the back part of the head and neck, so that the eye lies in front of the ear and below its level. The point where the back part of the head is continuous with the trunk is marked by a concavity, called the *Nackengrube*. In the fœtus eight weeks old, elevation of the greater part of the head has taken place, so that now the mid-brain lies above instead of anterior to the hind-brain, the eye lies in front of the ear, but more nearly at the same level, and the *Nackengrube* is almost obliterated. In the six weeks' embryo there are no traces of eyelids, the external ear is scarcely recognisable, and the maxillary processes have little more than united in the median line anteriorly; in the eight weeks' fœtus the eyelids are present, although

not fully formed, the concha is quite distinguishable, the anterior fusion of the maxillary processes is complete, and the face has taken on the human appearance (eyes, nose, mouth, chin). In the six weeks' embryo the upper limbs (in profile views) reach beyond the level of the heart, show the tripartite division, but are still strikingly bud-like; in the eight weeks' fœtus they reach beyond the anterior margin of the chest (in profile views), show clearly their three segments and five separate digits, and are flexed at the elbows and bent upwards towards the face. Similar but less marked changes take place in the lower limbs. The anterior contour of the trunk in the six weeks' embryo shows very evident bulging due to the presence of the heart and liver; this character is not so noticeable in the eight weeks' fœtus, although in it also the liver is of "relatively enormous dimensions," and reaches well into the hypogastric region. The epidermis at the end of the first month consists of two layers, and this two-layered stage lasts till the end of the neofœtal period; probably the outer layer of cells represents the epitrichium. The dermis is not yet differentiated into corium and subdermal layer; but the *anlage* of the mammary gland can be seen at the eighth week. The caudal projection (true tail) which attains its maximum about the thirty-fifth day (end of fifth week) becomes less and less marked during the neofœtal period, and has disappeared as a free appendage at the end of it (attainment of "human" form). During this eventful period, also, the protrusion of intestine into the umbilical cord increases to reach its maximum about seven and a half weeks; the genital tubercle which at first lies anterior to or within the orifice of the cloaca becomes more prominent, although it cannot yet be distinguished as penis or clitoris. Such are the external changes taking place in the organism during this transition time of neofœtal life, those most noteworthy being the elevation of the head, the disappearance of the tail, and the specialisation of the face and limbs.

The *internal* changes are no less wonderful and epoch-making. They are also numerous, and call for some kind of classification. They may be conveniently subdivided into—(1) the more marked or more typically embryonic changes, and (2) the less marked and more specially fœtal changes. In the former group, I place the changes which occur in the skeleton, in the cranium and its contents, and in the pelvis and lower part of the abdomen and their viscera. In the latter group may be ranged the changes, slight in character, which take place in the organs of the thorax and upper part of the abdomen.

1. *The skeletal changes.*—The changes which occur in the skeleton are chiefly of the nature of commencing ossification. Ossification begins in the neofœtal epoch, to end far on in post-natal life—a developmental change late of appearance, late also of completion. At the seventh week ossific nuclei appear in the clavicle (first bone, then, to become bone); in the shaft of femur and of tibia; in the frontal, parietal, interparietal, and, perhaps, in the squamosal and palatine bones; in the bodies of the vertebræ, at any rate in the dorsal region; and in the ribs (in this week or the next). In the eighth week the number of ossific nuclei is increased by the appearance of those for the shafts of the humerus, radius, and fibula; for the nasals, lachrymals, vomer, superior maxillaries, and malars; for most of the vertebræ; and possibly also for the metatarsals and metacarpals. Ossification then has made a commencement at the end of the second month of intra-uterine life. The rest of the skeleton, though not ossified, is already definitely mapped out in cartilage or membrane, *e.g.* the skeletal pieces of the limbs. It is noteworthy that the sternum consists of two cartilaginous lateral halves, still separate; and that the neural arches have not yet met on the dorsal side of the spinal cord. The condition of the spinal cord may be referred to here. It equals in length the vertebral column, the lumbar and cervical enlargements are indicated, the central canal begins to contract towards the close of the neofœtal period, and the anterior fissure begins to appear and the grey matter rapidly to increase. The notochord has begun to disappear.

2. *The cephalic changes.*—In the region of the face during the neofœtal period there are noteworthy changes. The nasal processes grow to form the external nose; the *anlage* of the lachrymal duct is present at the sixth week as a solid ridge; the development of the teeth begins with the formation of the dental groove and ridge at the seventh week, and the budding of the enamel organs at the eighth week. The *anlage* of the submaxillary gland is present at the beginning of neofœtal life, that of the sublingual appear soon after, and that of the parotid at the eighth week; about the same time chondrification of the larynx begins. Of all the internal changes in the head-end of the fœtus at this time, those of the brain are of most importance. The unequal growth of the various parts of the brain, which has already led to the production of mid-brain flexure and neck-bend, continues; the wonderful expansion of the cerebral hemispheres makes a commencement, and at the end of the period these structures have expanded to the edge of the mid-brain; the Sylvian fissure or fossa was evident at the fifth week, marking off the frontal from the temporal lobe, and in addition there can now be seen the *Bogenfurche* or callosal fissure, these two being total grooves or true folds of the brain; and the base of the

olfactory lobe is carried forward by this same cerebral hemispherical expansion. The axes of the eyes become parallel; and there is fusion of some of the tubercles which go to form the external ear. There are already indications of all the cranial nerves, but at this time the cavity which exists in the optic stalk begins to close.

3. *The pelvic changes.*—At the opposite or pelvic end of the fœtus important changes are also taking place. The Wolffian body reaches its maximum of development at the seventh week, and at the eighth begins to resorb; the kidney, which measures barely 2 mms. in length at the sixth week, is 2·5 mms. at the end of the neofœtal period, shows commencing lobulation, and in it Malphigian corpuscles begin to form. It is stated that a dilatation of the allantois to constitute the urinary bladder takes place, but the details of the development of this part of the urino-genital apparatus have not been yet ascertained. The testis is histologically distinguishable from the ovary at the sixth week by the smaller number of *Ureier* (primitive ova or ovic cells) in it. The fusion of the Müllerian ducts has begun at the eighth week. Sex, therefore, is already recognisable in the neofœtal period, albeit the distinguishing character is microscopical.

4. *The thoraco-abdominal changes.*—It is a remarkable fact that after the sixth week of intra-uterine life the organs of the thorax and upper part of the abdomen may be said to have completed their development; during the remaining thirty-four weeks they grow indeed, but show no changes in their construction till birth forces new functions upon them; some of them do not change even then. This is specially true of the heart and great vessels, for they change little, if at all, between the beginning of the neofœtal and the end of the fœtal period. In the circulating blood red cells (nucleated) are the most numerous, but the red plastids (non-nucleated) have begun to appear. The liver also is well developed, and grows enormously in size in the second month, and the gall-bladder is present. The spleen is quite recognisable. There is a slight change in the pancreas, but in its position only; it lies at first parallel to the long axis of the body, and later comes to be directed transversely. The asymmetry of the lungs is seen even at six weeks, and the lobes are marked off as branches. The typical form of the stomach is indicated at the fifth week, before, therefore, the beginning of the neofœtal epoch; and the villi and glands of the intestine have begun to develop at the second month, although the intestinal coils continue to elongate during fœtal life, and may not have taken up their permanent position and relations even at the time of birth. The development of the thymus gland from the entoderm of the third gill-cleft has begun. The two lateral *anlages* of the thyroid gland have united with the single median *anlage* at the seventh week; the ductus thyreoglossus may remain open till the eighth week; and at the same time the formation of hollow acini has commenced. In one detail, however, development in this region is incomplete: the separation between the pleural and abdominal cavities has not taken place in a two months' fœtus.

Not only are there changes, external and internal, in the embryo-fœtus during the neofœtal period, but there are also alterations in the fœtal appendages of very considerable importance. The organism lies in the sac formed by the decidual membranes, the reflexa being still distinct from the vera; the chorion is villous all over, but the villi in the region where the placenta is soon to form are larger than the others, and are already vascularised to a greater degree by the allantoic or umbilical vessels; the decidual membranes and chorion weigh together from 11 to 15 grms.; the liquor amnii is present in the amniotic cavity to the amount of 10 to 13 grms.; the umbilical vesicle has atrophied, but is still to be seen attached to the abdomen of the neofœtus by a thin cord, and doubtless there is still some circulation going on in the vitelline or omphalo-mesenteric vessels. As has been already stated, the projection of intestine into the umbilical cord is increased during the first week of the neofœtal period. The great changes seen in the environment of the fœtus at this epoch are the replacement of the vitelline by the allantoic or umbilical circulation, and the progressive growth in importance of the placental over the general chorionic circulation.

The end of the neofœtal period therefore coincides with the beginning of the placental connections. There is thus a sort of birth before birth, a transition not so sharp as that which occurs at the tenth month of intra-uterine life, but nevertheless definite enough and of great importance. Further, just as there are many traces of the fœtus to be seen in the new-born infant, so in the neofœtus there are not a few indications of the embryo; there are in it still some signs of typical embryonic or developmental activity, as the preceding paragraphs abundantly have demonstrated. By the end of the third month, as will be seen, the new-born fœtus is fairly established under the placental régime, its yolk-sac (vitelline) connections can be dispensed with and all circulatory activities can be concentrated in the allantoidal union with the decidua serotina. The transition thus accomplished is not without its element of danger; and just as the neonatal period is commonly one of danger to the new-born infant, so the neofœtal is full of risk to the "new-born fœtus." It is, at any rate, a fact well known that intra-uterine life is often brought to an untimely end by abortion at the third month.

The incidence of abortion so immediately after the neofœtal period suggests want of complete adaptation to the new condition of life, in other words, a defective establishment of the placental connections.

The new organism has now (end of second month) passed out of the distinctly formative or embryonic stage into the fœtal condition when growth rather than organ-formation is the predominant phenomenon; its embryology is over. As a fœtus it will be studied again, and its life, inside the uterus, will be followed up to the time when it is expelled into the world as a new-born infant or neonatus. These matters are dealt with in another article (Fœtus).

Embryoma.—A tumour made up of tissues derived from all the three layers of the blastoderm, and resembling, therefore, in this respect, an embryo (*Wilms*); a teratoma. Common examples of embryomata are found in the dermoid cysts of the ovary and testicle. *See* Tumours (*Dermoids*).

Embryonal or **Embryonic.**—Relating to or resembling an embryo. *See* Embryology; Fœtus and Ovum, Development of (*Embryonic Area*); Pregnancy, Intra-Uterine Diseases (*Embryonic Life*); Syringomyelia (*Embryonal glial tissue*); etc.

Embryotomy. — Comminuting operations performed upon the fœtus in order to reduce its bulk or the bulk of any of its parts (head, abdomen, etc.), and so facilitate its expulsion from, or extraction out of, the maternal passages. *See* Cleidotomy; Labour, Management (*Transverse Presentations, Embryotomy*); Labour, Operations (*Embryotomy*).

Embryulcia. *See* Embryotomy and *Cross References* thereunder.

Emergency Certificate. *See* Insanity, General Treatment (*Reduction of Function, Legal Provisions, Urgency Order*).

Emesis.—Vomiting (Gr. ἐμέω, I vomit). *See* Emetics.

Emetics. *See also* Alkaloids (*Apomorphina, Emetina*, etc.); Bronchi, Bronchitis (*Treatment, Emetics*); Lungs, Vascular Disorders (*Œdema, Treatment, Emesis*); Pharmacology; Physiology, Food and Digestion (*Vomiting*); Toxicology (*General Treatment of Poisoning, Emetics*); and under the various emetic drugs, such as *alum*, *ammonium carbonate*, *antimony*, *apomorphine*, *copper*, *ipecacuanha*, *mustard*, *senega*, *sodium chloride*, *squill*, and *sulphate of zinc*. These are agents that produce vomiting.—The act of vomiting is usually preceded by feelings of nausea, during which a copious supply of saliva is poured into the mouth. This being swallowed, carries down with it a certain quantity of air which subsequently helps in the discharge of the gastric contents, by assisting in the opening of the cardiac sphincter.

The mechanism of vomiting consists in a deep inspiratory effort being made, by which the diaphragm is pushed down as far as possible on to the stomach, the lower ribs being at the same time forcibly indrawn; the glottis is closed so that no air can enter the lungs, but probably some is swallowed into the stomach. This inspiratory action is followed by a sudden violent expiratory contraction of the abdominal muscles and diaphragm, and as the glottis is closed, this results in pressure on the abdominal contents, the cardiac orifice at the same time being opened by some contraction of its muscular fibres. The mouth is held widely open and the neck stretched, affording as straight a course for the vomit as possible.

There is an additional expiratory effort which serves to prevent the vomit passing into the larynx, the posterior pillars of the fauces being closed to prevent the contents passing into the nose. This is ineffective if the vomiting is severe.

Thus in vomiting there are two main factors, the extrinsic pressure of the abdominal walls on the stomach and the dilatation of the cardiac orifice. These are ineffectual unless they are both called into action. The former alone, however violent the effort may be, only produces *retching*. The intrinsic movements of the stomach, with an open cardiac orifice, are unable to do more than eject gas or water-brash (pyrosis). The two main factors do not usually come into play at once, hence the sensation of nausea is usually at first followed by retching, which becomes vomiting when the cardiac orifice is open.

During vomiting the pylorus is usually closed, and so prevents any of the stomach contents passing into the duodenum, but if the gall-bladder is full some of the bile may pass through the pylorus and find its way into the stomach (bilious vomiting).

The nervous mechanism involved in this act is governed by a centre situated in the medulla oblongata, and is capable of being stimulated by afferent impulses reaching it from many sources.

The more common sources of reflex stimulation are stimuli applied to the peripheral nerves, as tickling the fauces with a feather, irritation of the gastric mucous membrane, obstruction of the intestine, hernia, etc. Vomiting from renal and biliary calculi, and the morning sickness of pregnancy, are also apparently of reflex origin.

Lastly, the centre may be thrown into action by stimulation of parts of the brain higher up

than itself, as vomiting produced by smells, tastes, ideas, and in some cases from cerebral disease.

Drugs acting on the different organs, mouth, pharynx, œsophagus, stomach and intestines, on the biliary and renal passages, kidney, peritoneum and uterus, or on their centres, might be considered as emetics.

It is usual, however, in describing emetics only to consider those which do so either directly by acting on the stomach or on the centre in the medulla. The term *indirect emetic* is applied to those that produce vomiting by their action on the vomiting centre without being taken into the stomach. Such are ipecacuanha, apomorphine, tartar emetic, senegæ, and squill. The *direct emetic* is one that causes vomiting reflexly because they are applied to the stomach, *e.g.* the sulphates of zinc, copper, alum, carbonate of ammonium, salt, mustard, warm water, chamomile. Tickling the fauces with a feather may be relied upon to produce reflex vomiting either alone or in combination with a drug.

Some confusion has arisen between the terms direct and indirect emetics; they are, however, used to denote the connection between the stomach and the drug, and not the drug and the centre of vomiting.

Apomorphine, tartar emetic, and ipecacuanha are very powerful in their action, and are also very depressant.

Uses.—Emetics are firstly used to remove the contents of the stomach. If the organ is over-distended and there is a feeling of nausea, or if there is unsuitable food in the stomach, causing irritation and not undergoing proper digestion, as in some cases of dyspepsia and sick headache, a copious draught of lukewarm water, or of mustard and water, is usually all that is required. If the stomach contain a poison, it is well to get rid of it as soon as possible, and to prevent absorption, sulphate of zinc or copper, or mustard and water, are the best. If the poison should happen to be a powerful gastric intestinal irritant it is preferable to wash out the stomach rather than use an emetic.

Secondly, emetics can be used to aid the clearance of the air-passages in children who cannot expectorate well, as in bronchitis or laryngitis—the usual emetic being ipecacuanha; but in croup something with a quicker action is wanted, such as sulphate of copper or zinc, and tickling the back of the fauces. If there is much depression, carbonate of ammonium is best, as it is a stimulant as well as an emetic. An emetic may also be useful for the removal of a foreign body impacted in the larynx. So-called "biliousness" is more rapidly relieved by an emetic than by a purgative, as there is no chance of its absorption through the stomach. In ague, if there be much bilious vomiting, an emetic of ipecacuanha or hot water will clear the stomach and allow of the administration and retention of the quinine.

Caution.—Patients suffering from prolapse of uterus or rectum, peritonitis, hernia, aneurysm, and the like, are not suitable subjects for an emetic on account of the straining. For the same reason caution must be used in administering emetics to subjects with atheromatous vessels and high arterial tension, as the straining may lead to hæmorrhage.

Emetina or **Emetine.**—An alkaloid contained in ipecacuanha root. *See* Ipecacuanhæ Radix.

Emetomania.—A morbid desire or longing for the use of emetics; the converse of *emetophobia*, the morbid fear of emetics or emesis.

Emigration of Leucocytes.—One of the phenomena of inflammation, once considered to be the central phenomenon of that morbid process. Some time after the commencement of inflammation (two hours in the case of irritants applied to the web of the frog's foot) the leucocytes are seen to be gathering in the peripheral part of the blood stream in the vessels; they then attach themselves to the vessel-walls; and then by an amœboid movement they pass through the walls, which they penetrate by using the spaces between the endothelial cells. The leucocytes which chiefly emigrate are the polymorphonuclears; but others (the lymphocytes, the eosinophiles, and perhaps the large mononuclears) occasionally pass through. The vessels from which the emigration most often proceeds are the venules, but the capillaries occasionally show the same phenomenon, and the arterioles very rarely exhibit it. The cause of leucocyte emigration is probably to be found in *chemiotaxis* (*q.v.*), and the purpose of it is apparently to enable the leucocytes to act more efficaciously as phagocytes, by ingesting and (sometimes) digesting foreign substances such as bacteria.

Eminence or **Eminentia.**—Any prominence, projection, projecting ridge, or the like, such as the *bicipital* eminence on the radius (for the attachment of the biceps muscle), the *frontal* eminence (on the frontal bone), the *thenar* and *hypothenar* eminences (on the palm of the hand), the eminentiæ *mamillares* (or corpora albicantia), etc.

Emission.—Ejaculation or discharge of a secretion, *e.g.* of semen, as in spermatorrhœa, involuntary or nocturnal emissions, etc.

Emmenagogues.—*See also* Cantharides; Ecbolics; Ergot; Manganese; Menstruation and its Disorders (*Amenorrhœa*); Myrrh; Pharmacology; Purgatives, etc.—A drug which stimulates and regulates

the menstrual functions is called an emmenagogue.

The causes of disturbance of the menstrual functions are very varied, and will be found fully treated of under the heading "Menstruation." It is sufficient to mention here that when the general health is below par from nervous debility, anæmia, tuberculosis, an *indirect* emmenagogue is required, that is, one which will act by improving the general tone of the nervous system, or by increasing the quantity and quality of the blood. Such may also act by stimulating the adjacent organs, and thus reflexly acting on the uterus. The principal are purgatives, especially aloes in any form, and tonics—iron, arsenic, strychnine, potassium permanganate, cod-liver oil. Hot water and mustard, foot and hip baths, are also beneficial when menstruation is delayed.

Direct emmenagogues are those which exert a local action on the uterus. These are ergot, quinine, savine, guaiacum, hydrastis, rue, asafœtida, myrrh, cantharides, and borax. These, when given in moderate doses, gently stimulate the uterus, thus producing menstruation. If menstruation is profuse they check the excessive flow by toning up the uterus. When given in large doses they may produce abortion, and are then termed *ecbolics.* Their use in large doses is attended with very considerable risks.

Emmetropia.—The normal condition of the eye in respect to refraction; the principal focus is exactly the distance behind the crystalline lens at which the layer of rods and cones in the retina is situated, so that rays of light are focussed upon these rods and cones without any effort at accommodation. *See* ACCOMMODATION; ASTHENOPIA; PHYSIOLOGY, SENSES (*Vision, Formation of Pictures upon the Retina*); REFRACTION (*Emmetropia*); RETINOSCOPY (*Emmetropia*).

Emodin.—A constituent of rhubarb root, having the formula $C_{40}H_{30}D_{13}$ (or $C_{15}H_{10}O_5$); it is closely related to chrysophanic acid.

Emol.—A soapy mineral, with emollient properties, used in skin diseases.

Emollients.—Softening, relaxing, protecting, or lenitive substances, such as poultices, oils, fats, etc.; they have a softening, a moistening, or a warming effect, or else they simply form a coating on the skin which preserves it from injury, friction, or cracking. *See* PHARMACOLOGY.

Emotion. *See* CHOREA (*Etiology, Fright, Emotion*); HYSTERIA (*Convulsions, Second Period, Emotional Attitudes*); INSANITY, NATURE AND SYMPTOMS (*Index of Mental Functions, Feeling, Emotions, Affections*).

Empathema.—Ungovernable or uncontrollable passion, leading to perversion or subversion of reason and judgment.

Emphysema. *See* BRONCHI, BRONCHITIS, ACUTE (*Morbid Anatomy, Collapse of Lung and Compensatory Emphysema*); BRONCHITIS, CHRONIC, IN CHILDREN (*Prognosis, Emphysema*); CHEST, CLINICAL INVESTIGATION (*Alterations in Size and Shape, Emphysema*); CHEST, INJURIES OF (*Viscera, Wounds*); CHEST-WALL, AFFECTIONS (*Emphysema*); CONJUNCTIVA, DISEASES OF (*Emphysema*); EMPHYSEMA, SURGICAL; LUNGS, TUBERCULOSIS (*Complications, Integumentary, Cutaneous Emphysema*); LUNGS, EMPHYSEMA OF; MALINGERING (*Cutaneous Emphysema*); MEDIASTINUM (*Mediastinal Emphysema*); MOUTH, INJURIES AND DISEASES OF THE JAW (*Fracture of Superior Maxilla, Complications*); NECK, REGION OF (*Injuries, Surgical Emphysema*); OXYGEN (*Use in Pulmonary Emphysema*); PLEURA, AFFECTIONS OF, SURGICAL (*Injuries, Emphysema*); SCROTUM AND TESTICLE, DISEASES OF (*Emphysema of Scrotum*).

Emphysema, Surgical.—Emphysema is the term used to denote the presence of air in the cellular tissues of the body. In order to differentiate the condition from vesicular emphysema of the lungs, it is also called subcutaneous, cellular, or surgical emphysema. It is caused by—

1. Wounds of the lung, occasionally by a punctured wound, more frequently by a subcutaneous wound of the lung due to a fractured rib. It may be associated with pneumothorax, but as a rule this complication does not occur owing to cohesion of the visceral and parietal layers of the pleura. In either case, however, air is forced into the cellular tissue during expiration, and appears first in the chest-wall near to the seat of injury. In rare cases the cause may be disease of the lung, *e.g.* rupture of a vomica or pulmonary abscess.

2. Affections of other parts of the respiratory tract—wounds of the trachea, intubation, ulceration of a bronchus, or of the larynx. Fractures of the skull involving some of the accessory sinuses of the nose may be associated with emphysema, which is usually localised to certain regions, *e.g.* the orbit.

3. Wounds of the thorax involving the pleura, but not the lung. In rare cases air may be forced during expiration from the pleura into the cellular tissues.

4. Subpleural rupture of pulmonary vesicles by violent expiratory efforts, as in pertussis, less frequently in capillary bronchitis, diphtheria, or in violent straining during parturition.

5. Injury or disease of the alimentary tract—œsophagotomy, perforation of gastric ulcer, ulceration or rupture of the intestine, etc.

Symptoms.—The most common cause of subcutaneous emphysema is fracture of the ribs,

other causes being for the most part rare. The *appearances* produced are those of a swelling with ill-defined limits, and over which the skin appears healthy. The part is soft to the touch, and on slight pressure fine crepitation can be elicited, which is highly characteristic. The region where the air first makes its appearance subcutaneously is of importance in determining the cause. Thus the emphysema appears first in the chest-wall when due to a wound of the lung, but first in the flanks, and then on the front of the abdomen when due to rupture of the intestine. In subpleural rupture the air escapes into the interalveolar tissue (interstitial emphysema), and thence into the mediastinum, and appears first at the neck. The extent of the emphysema is very variable. In the majority of cases it is localised to the site of its initial appearance, in others it may thence spread. The infiltration of air is often limited by the attachments of fasciæ, and although general emphysema is of rare occurrence, the trunk, neck, and limbs may all be involved.

Treatment.—Pressure is useful in limiting the emphysema. Other measures must be adopted if indicated by the causal process, and the possibility of suppuration should be remembered especially if there be an external wound.

Emphysema must be clearly distinguished from the condition where gases in the tissues are due to the action of bacteria. Apart from the gangrenous emphysema due to the bacillus of malignant œdema, somewhat similar cases have been recorded by Fraenkel (B. phlegmones emphysematosæ), Wicklein (B. emphysematis maligni), etc., where the process was due to other anaerobic bacilli which are probably not all identical. Gas may also be found, associated with anaerobic bacilli, in the tissues post-mortem, usually, however, in the internal organs (German, Schaumorganen), and especially in the liver. The gas-forming bacillus found by Welch and Nuttal has been termed "Bacillus aerogenes capsulatus."

Empirical.—Based on experience and observation (Gr. ἔμπειρος, experienced), and not on scientific theory; empirical treatment may be successful, although the reason of its efficacy be unknown or inexplicable by philosophical or scientific methods of reasoning.

Emplastra.—Plasters, for external application; *Emplastrum plumbi*, or lead plaster, is the basis of nearly all the other official emplastra (*e.g.* Emplastrum hydrargyri, E. saponis, E. resinæ, E. belladonnæ, etc.).

Employment, Hours of.—According to Sanitary Law the employment of young persons (14 to 18 years) and women in textile factories must not exceed 12 hours a day, 5 days a week, and 6 hours on Saturdays; children must not be employed for more than half days, unless these be alternate days, and not on two successive Saturdays. In non-textile factories and workshops, women and young persons must not be employed longer than 12 hours on 5 days a week nor 8 hours on Saturdays. In both cases regular times are to be set apart for meals, and work must not be continuous for more than 4½ hours (in textile factories) or 5 hours (in non-textile factories) without at least half an hour for meals. "An occupier may not knowingly employ any woman or child in any factory or workshop within four weeks after she has given birth to a child."

Emprosthotonos.—The condition in which the whole body is curved, with the concavity anterior. *See* HYSTERIA (*Convulsions, First Period*); TETANUS (*Clinical Features, in Paroxysms*).

Emprosthozygosis.—Conjoined or united twins, in which the union is face to face, above the umbilicus (Gr. ἐμπρόσθιος, in front, and ζυγόω, I join together); the twins may be equally or unequally developed; under this term (introduced by Thompson Lowne) are included the xiphopagous and thoracopagous monsters and some parasitic types (*e.g.* that in which an acephalous twin is attached to the lower end of the sternum of its autosite). *See* TERATOLOGY.

Empyema. *See* ASPIRATOR, USES OF; BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Embolism, Causes*); BRONCHI, BRONCHIECTASIS (*Diagnosis, Prognosis*); DIAPHRAGM, SURGICAL AFFECTIONS OF (*Sub-phrenic Abscess*); JOINTS, DISEASES OF (*Empyema or Pus in the Joint*); KNEE-JOINT, DISEASES OF (*Cold Abscess or Empyema*); LIVER (*Tropical Abscess, Prognosis, Rupture*); LUNGS, GANGRENE (*Etiology*); NOSE, CHRONIC INFLAMMATION (*Atrophic Rhinitis, Diagnosis, Empyema of Antrum*); NOSE ACCESSORY SINUSES, INFLAMMATION (*Chronic Suppuration or "Latent Empyema"*); OSTEO-ARTHROPATHIES; PLEURA, DISEASES OF (*Empyema*); PLEURA, AFFECTIONS OF, SURGICAL (*Exploration of the Pleura, Empyema*; PNEUMONIA, CLINICAL (*Complications*).

Ems. *See* MINERAL WATERS (*Muriated Alkaline*).

Emulsification. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Pancreatic Secretion, Pialyn*).

Emulsin.—A ferment existing in bitter and sweet almonds; it is like casein; and it causes amygdalin ($C_{20}H_{27}NO_{11}$) to break up into benzaldehyde (C_6H_5COH), hydrocyanic acid (HCN), and glucose ($ZC_6H_{12}O_6$); synaptase.

Emulsion.—"A milky liquid, consisting of water holding in suspension minute particles

of oil or resin by the aid of some albuminous or gummy material" (*Sydenham Soc. Lexicon*); the liquid must have a high specific gravity; and when the particles consist of a heavy powder the result is called a *suspension* rather than an emulsion (e.g. *Lotis hydrargyri nigra*); mucilage of acacia is often used to form emulsions. *See* PRESCRIBING.

Emunctories.—The excretory organs (*e.g.* the kidneys); the nose (in sneezing) used to be called the "emunctory of the brain."

Enamel. *See* TEETH (*Anatomy*).

Enarthrosis.—A diarthrodial joint (such as the hip), freely movable in all directions; a ball and socket articulation.

Encanthis.—A tumour or swelling of the caruncle near to the inner canthus of the eye; it may be benign, malignant, or due to a concretion.

Enceinte.—Pregnant (Lat. *incincta*, ungirt).

Encephalitis. *See* BRAIN, INFLAMMATIONS (*Encephalitis, Acute and Chronic*); HEADACHE; PARALYSIS (*Cerebral Diplegia, Causation, Fœtal Encephalitis*).

Encephalocele.—A cephalocele containing part of the cerebrum or of the cerebellum; an illustration is given on Plate facing p. 78 of Vol. II. *See* BRAIN, SURGERY OF (*Cephalocele, Encephalocele*); HYDROCEPHALUS (*Complications*); LABOUR, FAULTS IN THE PASSENGER (*Malformations, Encephalocele*); ORBIT, DISEASES OF (*Tumours, Encephalocele*).

Encephaloid.—Resembling brain substance; usually applied to a variety of soft cancer.

Encephalopathy. — Brain - disease, especially when due to disease of some other part of the body or of a constitutional kind; it is commonly used in connection with the cerebral symptoms produced by lead-poisoning (*Saturnine encephalopathy*). *See* TRADES, DANGEROUS (*Lead-Poisoning, Symptomatology*).

Encephalotomy. *See* CEPHALOTOMY.

Enchondroma. — A neoplasm composed of cartilaginous tissue. *See* TUMOURS (*Connective Tissue Type, Chondromata*); LABOUR, PRECIPITATE AND PROLONGED (*Faults in the Passages, Pelvic Enchondromata*); MEDIASTINUM (*Mediastinal Growths, Enchondroma*); ORBIT, DISEASES OF (*Tumours, Solid, Enchondroma*); OVARIES, DISEASES OF (*Tumours, Enchondroma*).

Enclavement.—The embedding in the body cavities (during early antenatal life) of parts or derivatives of parts which are normally external; a theory of teratogenesis used to explain such developmental anomalies as dermoid cysts of the ovaries or of the brain. *See* TERATOLOGY.

Encystment.—The state of becoming or being enclosed in a sac or cyst, *e.g.* an encysted hernia (*vide* HERNIA, *Conditions of*), or encysted hydrocele (SCROTUM AND TESTICLE, DISEASES, *Hydrocele*).

Endarteritis. — Inflammation of the internal coat of an artery. *See* ANEURYSM (*Etiology, Chronic Arterial Disease, Endarteritis Deformans*); ARTERIES, DISEASES OF (*Endarteritis Deformans, Obliterative Endarteritis*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Hæmorrhage, Etiology*); INSANITY, PATHOLOGY OF (*Pathological Anatomy, Cerebral Blood-Vessels*); SPINAL CORD, MEDICAL (*Morbid Anatomy, Vascular Lesions*); STOMACH AND DUODENUM, DISEASES OF (*Ulceration, Morbid Anatomy*).

End-Bulb. *See* SKIN, ANATOMY AND PHYSIOLOGY (*Terminal Corpuscles, Tactile*).

Endemic.—The term applied to diseases which prevail in certain places, as opposed to *pandemic* diseases which exist everywhere. *See* EPIDEMIOLOGY (*Introductory, Definitions*); HÆMATURIA ("*Endemic*").

Endermic. Medication.—The application of drugs to the skin, either with the epidermis still present (by friction), or after it has been removed by means of a blister, when absorption is possible.

Endo-appendicitis. — Inflammation of the lining membrane of the appendix vermiformis.

Endocarditis. *See* HEART, MYOCARDIUM AND ENDOCARDIUM (*Endocarditis and Malignant Endocarditis*); BONE, DISEASES OF (*Acute Osteomyelitis, Complications*); CHOREA (*Etiology, Rheumatism and Endocarditis*); CHOROID, DISEASES OF (*Suppurative Choroiditis in Ulcerative Endocarditis*); LUNG, TUBERCULOSIS (*Complications, Endocarditis*); MALARIA (*Diagnosis, Ulcerative Endocarditis*); PNEUMONIA, CLINICAL (*Complications*); PREGNANCY, INTRA-UTERINE DISEASES (*Fœtal Endocarditis*); PUERPERIUM, PATHOLOGY (*Ulcerative Endocarditis*); PURPURA (*Symptomatic, Infectious, Malignant Endocarditis*); RHEUMATISM, ACUTE (*Symptoms and Course, Endocarditis*); RHEUMATISM IN CHILDREN (*Symptoms, Endocarditis*); SYPHILIS (*Tertiary, Pathology, Vascular System*); TYPHOID FEVER (*Diagnosis, Ulcerative Endocarditis*); URETHRA, DISEASES OF (*Gonorrhœa, Complications*).

Endocardium. *See* HEART, PHYSIOLOGY; PHYSIOLOGY, CIRCULATION (*Heart, Endocardium*).

Endocervicitis.—Inflammation of the mucous membrane of the cervix uteri. *See* UTERUS, INFLAMMATION OF (*Chronic Cervical Catarrh*).

Endocolpitis. — Inflammation of the vaginal mucous membrane. *See* VAGINA, DISORDERS OF (*Inflammatory Affections, Vaginitis*).

Endocyme. — That form of monster (double) in which one fœtus is contained within the other; fœtus in fœtu.

Endocyst.—The lining membrane of a hydatid cyst. *See* HYDATID DISEASE (*Development*).

Endoderm.—The hypoblast layer of the blastodermic vesicle. *See* FŒTUS AND OVUM, DEVELOPMENT OF.

Endo - Ferments. *See* ENZYMES (*Nature*).

Endognathion.—The inner segment of the intermaxillary bone. *See* PALATE, CONGENITAL MALFORMATIONS OF THE MOUTH (*Hare-lip, Development*).

Endolymph. *See* PHYSIOLOGY, NERVOUS SYSTEM (*Semi-circular Canals*).

Endometritis. *See* UTERUS, INFLAMMATIONS OF (*Endometritis, Acute and Chronic*); CURETTAGE; GYNECOLOGY, DIAGNOSIS; MENSTRUATION AND ITS DISORDERS (*Menorrhagia, Dysmenorrhœa*); PREGNANCY, OVUM AND DECIDUA (*Inflammation of Decidua*); PREGNANCY, DISEASES OF PLACENTA AND CORD (*Decidual Endometritis*); PREGNANCY, HÆMORRHAGE (*Detachment of Placenta, Etiology, Endometritis*); PUERPERIUM, PATHOLOGY (*Puerperal Infection, Endometritis*); STERILITY (*Etiology, Endometritis*).

Endophlebitis.—Inflammation of the inner coat of a vein.

End Organ. *See* PHYSIOLOGY, SENSES (*Tactile Sense, End-bulb, End-plate*).

Endosalpingitis. — Inflammation of the mucous membrane of the Fallopian tube. *See* FALLOPIAN TUBES (*Inflammation*).

Endoscope.—An instrument used for examining, by light, the interior of a hollow organ, such as the bladder, rectum, and (rarely) the uterus. *See* CYSTOSCOPE.

Endospores. *See* MICRO - ORGANISMS (*Hyphomycetes*).

Endothelioma. — A form of sarcomatous tumour in which the cells arise from the endothelium of lymphatics or blood-vessels; it may be met with in the ovary; angeiosarcoma or lymphangeiosarcoma. *See* PERITONEUM, TUMOURS OF (*Malignant Disease, Endotheliomata*); TUMOURS (*Endothelioma*); TUMOURS OF THE SKIN (*Endothelioma Cutis*).

Endothelium. *See* PHYSIOLOGY, TISSUES (*Connective Tissues, Endothelium*).

Endotrypsins.—Intra-cellular enzymes which dissolve albumin. *See* ENZYMES.

End Plate.—The expansion at the end of a motor nerve where it terminates in a striated muscle.

End Products.—The final results of metabolic processes in the body, thus arginin and lysin are regarded as the end products of a pancreatic digestion.

Endyma.—The epithelial lining of the cerebral ventricles.

Enemata.

See also CONSTIPATION (*Treatment*); CHOLERA, EPIDEMIC (*Treatment*); GASTRO-INTESTINAL DISORDERS OF INFANCY (*Constipation*); INVALID FEEDING (*Rectal or Nutrient Enemata*).

INTRODUCTORY.—Enemata are liquid preparations used for injection into the rectum. They are usually classified into three groups—nutrient, purgative, and medicinal, according to their action. Nutrient and medicinal enemata being intended for absorption, must be small in amount and non-irritating to the neuro-muscular mechanism of the intestine. Purgative enemata, on the other hand, being administered for the purpose of evacuating the bowel, must have the power of stimulating the intestinal muscle nervous mechanism, and also the glandular structures in the intestine. Hence the main feature of a purgative enema is its bulk, which is usually, for an adult, one, two, or more pints, while the bulk of a nutrient enema should not exceed four ounces.

MODES OF ADMINISTRATION.—Enemas are best administered with the patient lying on the side with the knees drawn up. The process of injection must be carried on slowly, with occasional pauses, and after the injection a towel may be pressed against the anus, otherwise there is a risk of premature contraction of the bowel, with expulsion of the enema. In children a certain amount of pressure exerted on the sides of the anus for a few minutes helps its retention.

When it is desirable to inject a large quantity the patient should lie first on the left side, then on the back, and lastly on the right side, so as to promote the filling of the intestine.

Cautions.—(1) In all cases avoid injecting air. (2) Always see that the end of the nozzle

is free and not pushed against the internal sphincter, the sacrum, or a mass of fæces. (3) Keep in mind the possibility of a syncopal attack following its use.

Apparatus.—Various forms of syringes are used for the injections. In cases where a *medicinal* agent is being given, and the quantity is about 2 to 4 oz., a small glass piston syringe with a slightly curved vulcanite nozzle is all that is required. For a *nutrient enema*, the quantity not exceeding 3 oz., a brass piston syringe or a simple elastic bottle with an ivory or gum-elastic nozzle can be used. Where a *purgative enema* is required, and the quantity is large, an instrument of the nature of a Higginson's syringe is most serviceable, care being taken to see that it is full before commencing the injection so as to avoid the injection of air. Each contraction of the ball injects on an average one ounce of fluid. For *irrigation of the bowel* an ordinary douche apparatus, as used in obstetric practice, with a large-sized rubber catheter at the end of the tube, is required. The patient is placed in the following position, with mackintosh sheet below, and draining into a pail. The douche can is placed 4 or 5 feet above the bed. The catheter is oiled and placed within the anus before the water is turned on. As the water flows the catheter is passed steadily upwards for 12 to 14 inches if possible. Plain water, or a 75 per cent solution of salt and water (one teaspoonful to the pint), seems to be as efficacious as any of the other fluids that are credited with antiseptic properties.

Therapeutic Uses. — *Constipation.* — The amount required for an adult is usually 1 to 2 pints; for a child of four years old, 4 to 6 ounces; and for an infant, 1 ounce. It may consist of soap and water, or soap and water with 3 or 4 ounces of turpentine, or gruel water either alone or mixed with olive or castor oil. These should all be given at a temperature of about 100° F. The enema magnesii sulphatis ℥xvj., and the enema aloes ℥x. (B.P.), are also valuable. Small enemas of cold water are very useful in some cases, probably acting by stimulating peristalsis.

Glycerine.—This acts both as a stimulant and irritant to the rectum, an injection of ʒj. or ʒij. producing a motion in about half an hour. In some cases glycerine produces a severe burning sensation, and can be advantageously diluted with equal parts of water or castor oil.

Glycerine suppositories, of various forms and sizes, are a convenient form for administration.

Intestinal obstruction.—The introduction of a large volume of soap, water, and oil in the manner previously described under *intestinal irrigation* may be very useful. In all cases of this nature, it should be borne in mind, large enemata should only be used after a very careful consideration of each individual case.

Diarrhœa and Dysentery.—The enema under these conditions may be desired to act either as an astringent, an anti-spasmodic, or a sedative.

For a sedative enema, the enema opii ℥ij., whose basis is mucilage of starch, is probably the best. For a child of one year, ♏ij. to ♏iij. of tinct. opii in ℥ss. of starch, of a suitable consistence, is sufficient.

As a sedative and astringent to the mucous membrane of the lower bowel, bismuth subnitrate (ʒj. in ℥iv. to ℥vj. of water) or mucilage of starch is useful.

If there is ulceration with a mucous discharge, silver nitrate (5 grs. to 1 pint water), zinc sulphate or copper sulphate (1 grain to the ounce of water), give better results.

In the summer diarrhœa of infants, tannic acid (10 to 30 grs. to a pint of water) appears to render inert some of the animal poisons present.

Irrigation may also have a soothing influence on the mucous membrane, and is useful in various forms of diarrhœa and in cases of recurrent colic.

In *dysentery* 4 to 6 pints of warm water, or of milk and water, have been injected as a form of internal fomentation.

If there is reason to believe that gangrene is present, and is chiefly located in the rectum, emollient and antiseptic enemata are indicated. The amount injected should never be sufficient to distend the bowel immoderately, or to cause pain. Infusion of linseed is good as an emollient and sedative enema. Enemata of warm water, with potassium permanganate, or a weak solution of carbolic acid, are of service when there are black sloughs or offensive debris in the stools.

In cases of *gangrenous rectitis* repeated enemata of water acidulated with lemon juice have been recommended.

Parasites.—The thread worm, or ascarides, can be got rid of by the following injections:—Enemata of salt and water, or lime water; spirit of turpentine, ʒij. to ʒiv. with a yolk of an egg in ℥iv. of water; infusion of quassia, ℥ij. to a pint of water; enema aloes, or asafœtida (B.P.), ʒiv. may also be employed.

In giving an enema of quassia it is well to remember that symptoms of poisoning have occasionally arisen, a condition of powerful narcosis for many hours, with respiration and heart's action extremely feeble, having been observed.

Hæmorrhoids.—When necessary nothing is better than a simple enema of ½ pint of cold water injected after breakfast. Slight bleeding from piles may be checked with injections of cold water.

Hæmorrhage.—After a profuse post-partum hæmorrhage the collapse is greatly assisted by the slow injection of one or two pints of warm saline solution, or even warm water, into the

rectum. The general condition of the patient rapidly improves, as the fluid is quickly absorbed by the rectum. The same form of enema given after a severe operation is of great help in allaying thirst.

Nutrient Enemata.—The following points must be borne in mind:—(i.) The absorbent powers of the mucous membrane of the rectum are slight and slow in action; (ii.) irritation may easily be set up. The food, therefore, must be easily absorbed, bland, and of small bulk. The ingredients and methods of peptonising and pancreatising will be described in a later article ("Invalid Feeding"). There are rectal suppositories of predigested foods in the market, and they are very useful at times.

It is sufficient to say here that the quantity should not exceed from 2 to 4 ounces, that the irritation of the rectum with its obstinate diarrhœa may be delayed by the cleansing of the rectum with a small injection of saline solution before each enema is given, and the administration of ♏v. tinct. opii in every second or third enema.

Nutrient enemata should not be given oftener than one every six hours. A skilful nurse can do a great deal in the successful maintenance of rectal feeding for a prolonged period. These forms of enemata are of little use in childhood from the difficulty of retention.

Medicinal agents, such as morphia and belladonna, are administered more conveniently by the rectum in the form of suppositories. Chloral may be administered in cases of eclampsia, 30 grains at the onset of the attack repeated every two hours until the fits stop. It is not advisable to exceed 3½ drachms in the twenty-four hours.

Enemata as a Diagnostic.—The diagnostic value of a purgative enema is considerable, and will be referred to in other articles, *e.g.* constipation, intestinal obstruction. It will suffice here to emphasise the fact that the use of a large purgative enema, sometimes repeated two or three times, has not infrequently indicated the simple nature of a case, when the symptoms had been such as to suggest much greater mischief, *e.g.* typhoid fever, tuberculous meningitis, intestinal obstruction from other causes, simple mania, etc.

As previously stated, careful deliberation is frequently required before deciding on the use of large enemata.

Enentery.—A variety of the teratological state known as omphalocephaly, in which a part of the embryo is abnormally lodged in the alimentary canal, either anteriorly (*omphalocephaly* proper) or posteriorly (*ourentery*); it has been noted in chicks artificially hatched, but not yet in mammals.

Energy. *See* Physiology, Food and Digestion (*Food-Stuffs, Energy-Values*); Physiology, Dietetics (*Energy-Requirements*).

Engadine. *See* Therapeutics, Health Resorts (*Switzerland*).

Engagement.—The first stage in the descent of the head of the fœtus into the pelvis (through the pelvic brim) in labour. *See* Labour, Physiology.

England. *See* Therapeutics, Health Resorts (*English*).

English Cholera.—Choleraic diarrhœa. *See* Cholera Nostras.

English Disease.—Rickets (according to early German writers).

English Malady.—Hypochondriasis.

Engorgement.—Over-distension (*e.g.* of the vessels or heart) with blood; congestion.

Enomania.—Delirium tremens. *See* Alcoholism.

Enophthalmus.—Morbid retraction of the eyeball. *See* Eye, Clinical Examination (*Introductory*).

Enosomania.—The form of insanity in which the patient believes that he has committed the unpardonable sin.

Ensiform.—Sword-shaped or ensal, *e.g.* ensiform cartilage.

Ensilage.—The process of keeping hay by placing it in trenches (silos) in the ground; its own weight is usually sufficient to compress; it has practically the same value as a food-stuff as the material (grass, maize, etc.) from which it is made.

Enteræmia.—Intestinal congestion.

Enteralgia.—Intestinal pain. *See* Appendix Vermiformis (*Appendicitis, Diagnosis*); Gout (*Irregular, Alimentary System*); Hysteria (*Sensory Disorders, Hyperæsthesia*).

Enterectomy.—Resection of a part of the intestine. *See* Abdomen, Injuries of (*Treatment*).

Enterepiplocele.—A hernia whose contents include omentum as well as intestine. *See* Hernia.

Enteric Fever. *See* Typhoid Fever; *see also* Alopecia (*Premature, after Enteric Fever*); Ankle-Joint, Diseases of (*Synovitis, after Enteric Fever*); Cholera Nostras (*Diagnosis*); Colon, Diseases of (*Simple Colitis, Diagnosis*); Diphtheria (*Diagnosis*); Heart, Myocardium and Endocardium (*Malignant Endocarditis, Diagnosis*); Hydropathy (*Acute Diseases, Enteric Fever*); Meteorology (*Enteric Fever in Autumn*); Milk (*Therapeutic, Patho-*

logical); Parotid Gland, Disorders of (*Parotitis in Enteric Fever*); Pregnancy, Affections and Complications (*Specific Fevers, Enteric*); Puerperium, Pathology (*Septicæmia, Diagnosis*); Therapeutics, Serum Therapy (*Anti-enteric Serum*); Thyroid Gland, Medical (*Acute Thyroiditis in Enteric Fever*); Undulant Fever (*Diagnosis*).

Enteritis. *See* Intestines, Diseases of (*Enteritis*); *see also* Gastro-Intestinal Disorders of Infancy (*Inflammatory Diarrhœa*); Peritoneum, Acute Peritonitis, General (*Diagnosis*); Typhoid Fever (*Diagnosis from Gastro-Enteritis*).

Enterocele.—An intestinal hernia. *See* Groin, Diseases of; Hernia (*Structures, Contents*); Pelvis, Perineum and Pelvic Floor (*Vaginal Enterocele*).

Enterocleisis.—Intestinal occlusion.

Enteroclysis.—Intestinal irrigation by means of large enemata (*q.v.*), as given in cholera, for instance.

Entero-colitis.—Inflammatory diarrhœa, or ileo-colitis. *See* Colon, Diseases of (*Ulcerative Colitis*); Intestines, Diseases of (*Enteritis*).

Enterocolostomy.—The operation of anastomosing the ileum to the sigmoid flexure; it has been proposed in cases of obstinate constipation (!).

Enterocystocele.—A hernia containing intestine and urinary bladder.

Enterocystoma.—A tumour of a persistent part of the omphalo-mesenteric duct.

Entero-epiplocele. *See* Enterepiplocele; Hernia (*Structures, Contents*).

Entero-hæmal Circulation.—The circulation between the blood and the alimentary canal, consisting in the pouring out of various secretions into the intestine and their partial re-absorption. *See* Physiology, Food and Digestion (*Fate of Digestive Secretions*).

Enterokinase.—A substance (zymin) in the intestinal secretion, which, acting on trypsinogen, converts it into active trypsin. *See* Physiology, Food and Digestion (*Pancreatic Secretion*).

Entero-lithiasis.—The formation of concretions (*Enteroliths*) within the lumen of the bowel. *See* Stools, Intestinal Sand.

Enteromenia.—Vicarious menstruation (or xenomenia) from the intestines; a doubtful occurrence.

Enteromyiasis.—Intestinal disease caused by the larvæ of flies.

Enteropexy.—The operation of fixing the intestine (by stitches) to the abdominal wall to keep it in position.

Enteroptosis. *See also* Chest, Deformities of (*Mechanical Causes, Enteroptosis*); Kidney, Surgical Affections (*Movable and Floating Kidney, Condition of Abdominal Organs*); Liver, Diseases of (Hepatoptosis); Stomach, Surgical Affections (*Gastroptosis*).

Syn.—*Glénard's disease, Visceroptosis* (Eccles), *Splanchnoptosis* (Stiller).

Definition.—In the sense used by Glénard, enteroptosis implies downward displacement of the stomach, transverse colon, and generally of the right kidney, accompanied by digestive disturbances and often by neurasthenic symptoms.

As employed by later writers, especially those of the German school, enteroptosis is rather applied to displacements of at least two abdominal organs (Schwerdt), without any distinction as to them individually. Nor does it appear that they attach so much importance to the dyspeptic or the neurotic accompaniments as the followers of Glénard.

Owing to the utter state of confusion into which the subject has drifted, and the conflict of ideas as to what really constitutes enteroptosis and what does not, any connected description of the matter is rendered hopeless and impossible.

The confusion into which the subject has fallen is entirely due to the unscientific and illogical application of the term enteroptosis to a somewhat fantastic and arbitrary group of symptoms and signs raised by Glénard to the dignity of individuality, thus elevating them to the rank of a special disease, whereas these signs and symptoms may be occasioned by other combinations of visceral displacement, or depend upon changes in position of one or two of the three organs included by Glénard, not necessarily of all.

Literally implying a falling down of the bowel, the term should be applied only to this. In default, however, of a more suitable name it will be convenient to consider it to mean the displacement downwards of any abdominal organ, associated with a similar descent of at least one other (Schwerdt), and accompanied by symptoms of disturbed digestion, with or without neurotic conditions.

The mere physical condition of ptosis of any abdominal viscus need not necessarily lead to functional error, indeed, in numbers of women visceral ptoses of greater or less degree lead to no ill result. It is only when descent of abdominal viscera causes or has been caused by pathological lesions, symptomatically expressed, that the expression enteroptosis or any of its component units become applicable.

Thus the relationship between enteroptosis as a medical entity and the displacement downwards of the various abdominal organs individually

remains a *quæstio vexata.* Any conclusion as to whether a floating kidney, a wandering spleen, a fallen stomach, or a movable liver, individually or in combination, really constitute an incomplete form of enteroptosis or not must be left for future systematism to determine. It would conduce to ease of understanding if the word splanchnoptosis were to be used to cover multiple displacements of abdominal organs, enteroptosis being confined only to intestinal prolapse, and the other special names to the dislocation of the several organs.

Etiology.—Many have been the theories advanced to explain the causes of enteroptosis, theories which contradict one another in almost every particular.

Owing to the uncertainty attaching to the actual lesions which are included under the term enteroptosis, any attempt to portray the etiology of the disease as an entity presents great difficulties to the writer. Glénard's disease, as interpreted by himself, would seem to depend upon weakness of the ligamentous bands suspending the several organs concerned to the abdominal parietes.

Glénard, in his original description, pointed out that the greater length of the intestinal tract compared with the same measurement of the cavity housing it—about ten times as long—necessitates arrangement of it in coils,—coils suspended by a series of ligamentous attachments to the posterior wall of the cavity. At each point where a coil bends to join the loop above or below it a sharper curve is formed. These points are particularly apt to hinder the passage of the intestinal contents, and if the bowel fall at one point its traction upon the next of these *points d'appui* will serve to drag, and possibly stretch, the ligaments, with the result that further descent is rendered probable.

A downward displacement of the colon and small intestine is logically followed by descent of the stomach, with or without antecedent signs of dilatation, as the removal of support from the stomach given it under normal conditions by the bowels, aided by the elastic pressure of the abdominal walls and of the atmosphere outside them, causes a strain upon the gastro-duodenal ligament which it is unable to bear unaided.

A dilated stomach of pronounced character, where stasis of the contents occurs and large accumulations of material gather, favours displacement by its size, and tends to cause descent of other organs.

The gastro-duodenal ligament or small omentum is not invested with any great power of resistance, and the same is true of the meso-colon. Dislocation of the organs supported by these membranes is therefore not uncommon, nor surprising, and the descent of the one generally implies falling of the other.

The downward displacement of the whole stomach involves stretching of the œsophagus and its attachments. The condition known as pyloroptosis results when the pyloric portion of the stomach descends without change of position of the cardia.

Glénard claimed a constant connection between one or other displacement of individual organs and a general tendency towards abdominal descent, a "ptosical diathesis." There is abundant evidence to prove that falling-down of the stomach as a whole, or of the transverse colon, is prone to lead to dislocation in other organs; and that the cause of one organ's descent may very well account for displacement of others; but equally clear evidence can be given as to the actual occurrence of ptosis in single organs unaccompanied by any general displacement, at least to any noteworthy extent, as, for example, in floating kidney.

Again, enteroptosis *per se* may follow other displacements, and is by no means invariably attended by signs or symptoms of disease, although several authorities assert the contrary opinion.

Still there are some who attribute enteroptosis to congenital predisposition. Stiller, for instance, describes the condition as one in which "downward displacement of the stomach, small intestine, the right or both kidneys, the liver occasionally, and the spleen still less frequently, occurs owing to weakening of the peritoneal folds." "It arises from an inborn, probably inherited predisposition, which largely depends upon weakness of the central and digestive nerve systems."

Stiller further believes that the mobility of the tenth rib varies with the degree of neurasthenic dyspepsia present, and consequently with the displacement of the abdominal viscera accompanying it. Nervous dyspepsia and enteroptosis are seldom seen apart from one another.

Although Stiller disregards the effects produced by tight lacing on the stomach, there is little doubt that the unnatural conditions caused thereby are capable of originating enteroptosis with or without nervous habit or hereditary slackness. Nervous dyspepsia may arise from antecedent enteroptosis; enteroptosis cannot be the offspring of nervous dyspepsia (Herzog).

Kelling supposed that a direct connection existed between the change in position of the stomach, the proportion of hæmoglobin in the blood, and the condition of bodily nutrition; but Küttner and Dyer, in 18 healthy subjects, whose ages ranged from 10 to 70 years, with a mean of $30\frac{1}{4}$, found the percentage of hæmoglobin to be 81·1 (50-95), while 18 similar subjects presenting signs of gastroptosis, with 33·3 years as their mean age, gave 81·8 per cent of hæmoglobin (62-90), no diminution of hæmoglobin had occurred.

Perhaps it may be postulated, from the more

recent writings on the subject, that the most probable causes of enteroptosis, or of the displacement of individual abdominal viscera, are loss of abdominal fat, artificial compression of the waist, pregnancy, weakened support by visceral tension, a slackened state of the muscles of the anterior and lateral abdominal parietes from various causes, and perhaps in a few cases congenital weakness of the supporting ligaments.

Dilatation of the stomach can often be traced to long-continued habits of overloading that organ with food and drinks; but such can hardly cause gastroptosis. The organ dilates because of a too frequent physical distension, which in itself tends to keep the upper part of the stomach in place by reason of the consequent increased intra-abdominal tension. Unless the tension common to all the abdominal contents be irregularly or inefficiently maintained no change in the positions of the contained organs can take place, unless weakness of attachments and deficient resisting power of the diaphragm permit of sinkage of organs, with occupation of their former places by other parts.

Etiological Factors

Author.	Factors.	Organs.
1. Glénard.	Weakness of peritoneal ligaments; sinking of bowel at ends of its transverse coils; a "ptosical diathesis."	Stomach. Transverse colon, right kidney. (Neurosis and dyspepsia.)
2. Stiller.	Weakness of peritoneal folds; inherited predisposition; weak nerve systems, central and digestive.	Stomach, small intestine, right or both kidneys, the liver, and the spleen rarely; movable tenth rib.
3. Obrastow.	Heredity; loss of tone of abdominal muscles; loss of peritoneal fat; neurasthenia.	
4. Schwerdt.	Atony of nervous system; corsets, leading to loss of tone in walls and impaired digestion.	Any two abdominal organs.
5. Symons Eccles.	Loss of abdominal tension.	General enteroptosis. "All viscera apparently hanging down in a pendulous belly."

Position of the Abdominal Organs.—No exact statements are possible as to the normal positions and limits of abdominal organs in the majority of persons. They vary markedly with the form of the chest, the state of nutrition generally, the fulness or emptiness of the stomach or bowels, in both sexes: in the female their relations depend also upon the extent to which compression of the waist has been carried, through the use of corsets, and upon former pregnancies.

The question is complicated by the fact that the hollow viscera displaced may also be dilated; indeed, a stomach may be displaced downwards while so dilated that its misplacement is obscured by the physical signs afforded by it. The normal stomach should not extend below an inch above the umbilicus when distended, or, at most, below the umbilicus; above, it should yield evidence by its resonant note of its lesser curvature being opposed to the left lobe of the liver; the fundus corresponding to the level of the sixth or seventh costal cartilage. No stomach can be ptosed if the fundus reaches as high as this.

Statistics.—Glénard diagnosed the presence of some degree of enteroptosis in 404 out of 1310 patients examined for dyspeptic troubles, or a proportion of 30·8 per cent. Of the 404, 306 were females. Einhorn gives the following figures:—

Patients Examined.	Percentage with Enteroptosis.
Males	6·2
Females	34·8
Both sexes	17·6

Huber found displacements of abdominal organs at 90 per cent of 50 female patients observed. Examination of the position of the stomach in 100 men, and the same number of both women and children, by Küttner and Dyer, afforded evidence of the presence of gastroptosis in 39 adult females, in 3 girls, and in only 4 males. Ten of the females were nulliparæ, 12 had borne one, and 17 more than one child. Hertz could only satisfy himself that in 5 out of 50 women examined the abdominal organs were in normal position, while Kellogg among 250 female patients in a lock ward, detected prolapse of the stomach and bowel in 232, or 92·8 per cent. Meinert examined 29 girls before puberty, and recognised the symptoms of gastroptosis in 28. Eccles, out of 468 dyspeptics personally examined, found 27 only with simple gastroptosis—8 in males (æt. 32-61), 19 in females (æt. 9-45).

Habel's figures as to the frequency of floating kidney in patients suffering from various diseases are interesting in this connection. Among 68 cases of locomotor ataxia—44 males, 24 females—he detected a movable kidney in 6, all females, or 25 per cent of their number. Of 5393 female patients examined, 53 presented signs of this lesion, or about 1 per cent, while of 16,518 males, in only 8 could such a diagnosis be established, or 0·048 per cent; the totals yielding 21,911 patients and 61 examples of the lesion, or 0·27 per cent. Habel's figures contrast markedly with those of Glénard and Einhorn, even although they include other than dyspeptic cases; and also with Hertz's and Huber's more analogous series. The interpretation of enteroptosis by the first two observers implies the presence of movable kidney in their positive cases, and in the cases of the other two, the percentage arrived at for displacements generally implies a considerable proportion of renal dislocations.

Symptoms.—Objective.—Mathieu describes two forms of enteroptosis; the first, or "enteroptosis from outward" causes, showing characteristic alterations in the external shape of the

anterior abdominal wall; the second, "enteroptosis from inside," exhibiting no visible signs. The objective symptoms in the first form consist in flattening of the abdominal wall over the higher portion, with prominence of the lower. In thin subjects, if the stomach be full of food or gas, its entire outline may be visible, the curve of the lesser curvature showing below the xiphi-sternum, sometimes even reaching midway between this point and the umbilicus. The alternate inflation of the stomach and bowel with air, as Ewald recommends, usually demonstrates clearly the existence of this condition. Percussion over Traube's semilunar area yields a dull note whether the stomach be full or empty, while the lower margin of the liver can be seen or felt below its normal level, and the pancreas in some cases recognised as a transverse cord, a cord mistaken by Glénard for the transverse colon, an error, that is, according to Ewald. Splashing sounds can be elicited by more or less forcible percussion below the normal limit, and may be difficult to produce, or be absent, in the epigastric region. The gastric and intestinal movements induced by the action of the diaphragm are less closely associated with it than in health. In cases of pronounced gastroptosis the stomach invariably is deflected towards the left in addition to its downward displacement.

Forcible percussion of the abdominal wall over a displaced stomach within three or four hours of a meal produces splashing sounds at or below the level of the umbilicus, varying with the extent of the ptosis, and if inducible at a later period after the last meal, suggests dilatation as well as change of position.

Auscultation over the anterior abdominal walls while the patient drinks some water may afford evidence of the altered position of the stomach.

No correspondence can be traced between the chemical factors found on analysis of the gastric contents and enteroptosis. The contents vary, as in all nervous examples of dyspepsia; in one free hydrochloric acid is absent, in another normal, in a third excessive. Huber found in twenty-eight cases of floating kidney in females accompanied by symptoms of dyspepsia, and closely akin to those of enteroptosis, the absence of this acid in a free state in seven (25 per cent), normal in eleven (39·4 per cent), excessive in ten (35·6 per cent).

Huber describes the symptoms as closely resembling those of nervous dyspepsia, epigastric pain, more or less dependent upon food, heartburn, eructations, capricious appetite, constipation, headache, and insomnia. His cases were chiefly diagnosed as movable kidney, but his description applies also to enteroptosis.

It must be borne in mind that the displaced stomach and colon may also be the subjects of various degrees of dilatation,—conditions, indeed, which probably have had something to do with the causation of the altered intra-abdominal arrangement of organs.

Diagnosis.—The diagnosis of the conditions which go to make up the various forms of enteroptosis is easily arrived at, once the nature of the disorder has been grasped and is borne in mind. The commonly pendulous belly, absence of resonance in Traube's area, a stomach note below the level of health, associated with digestive troubles, and, it may often be, with neurasthenic manifestations, absence of the right kidney from its appointed place, lowered level of liver dulness, all conspicuously aid the physician. In doubtful cases the use of Einhorn's gastrodiaphanoscope may give one absolute proof; or the determination of the outline of the stomach by percussion alone, auscultatory percussion, or succussion with auscultation both in the erect position and after the patient has been placed in a recumbent posture, with his feet and pelvis raised and his head and shoulders lowered. Introduction of water in quantity into the stomach or into the colon *per rectum* is of greater service in diagnosis than the inflation of these organs with gas, because the weight of the fluid introduced, the patient meanwhile maintaining an erect position, tends to increase the downward displacement, while inflation with gas is rather apt to cause expansion laterally and upwards, and thus to obscure more than help elucidation.

Enteroptosis and gastroptosis are so intimately related to displacements of other abdominal viscera, and, in turn, symptoms of derangement of the stomach and bowel associated with obviously neurotic signs are so often the accompaniments of visceral errors in position, that a thorough physical examination of the abdomen and its integral parts should never be omitted in any case presenting symptoms approaching these characteristics It should be borne in mind that displaced organs may sometimes be detectable, which are by no means guilty of symptoms present and generally associated with them, for displacement does not invariably cause distress; and dilatation and paralysis of the sigmoid flexure, or of parts of the colon, with localised stases of the intestinal contents, may produce very similar results.

Prognosis.—The prognosis depends almost entirely upon the patient himself. If he consent to undergo a somewhat exasperating course of treatment in a proper spirit, observe the rules as to diet and habits laid down for him during a greater or less length of time (varying in each case), and be made to understand the precautions he must always take in the future against recurrence of the lesion, there is a great chance that he may recover from the symptoms and remain free from them. The condition is not exactly curable, but can be removed, and its recurrence completely guarded against, if the patient do what is asked of him.

Treatment. — The treatment of enteroptosis can merely be rational. The displaced organs must as far as possible be supported from without; best done through the application of a belt round the abdomen in such a way that the pressure exerted is greater below than above. The nature of the material used depends on the subjects treated; some find one kind irritating, some are rendered uncomfortable by others. The material used is immaterial, provided it be not of india-rubber (for this soon loses its elasticity), if it suffices to maintain the requisite pressure without inconvenience. Massage of the abdomen, fractional dieting, rest in a recumbent posture, or with the legs and pelvis raised, as Eccles has shown, and irrigation of the rectum and sigmoid, are all measures which may prove efficacious.

Günzburg has recommended the administration of fresh yeast equal to either a pea or a bean in size, thrice daily, so as to generate gas in the stomach, inflate that organ, thereby fixing its position, and, by its increase in size, helping to support the other viscera, until what time they have acquired a lessened proclivity towards change of position. This method of treatment is not applicable if the stomach be dilated as well as ptosed.

Symons Eccles has found the use of apparatus designed to keep the patient at rest in an inclined position, with the head low, the feet high, of service.

Surgical Treatment.—Stengel and Beyea have obtained good results by so suturing the gastro-hepatic omental attachments and the gastro-phrenic ligament that they were shortened.

Enterorrhaphy. — The closure (by sutures) of an intestinal wound, or the fixation of the intestine to another structure.

Enterorrhexis. — Intestinal laceration or rupture.

Enterospasm.—A purposeless tetanic contraction of the intestine whereby it is converted into a hard cord, affecting the colon more often than the small intestine, and apt to be confounded with appendicitis. *See* H. P. Hawkins' paper on the "Reality of Enterospasm and its Mimicry of Appendicitis" (*Brit. Med. Journal*, vol. i. for 1906, p. 65).

Enterostomy.—The making of a permanent opening into the intestine (*e.g.* in cases of stricture). The word *enterotomy* is sometimes restricted to the cases in which the opening is temporary.

Entoderm. *See* Endoderm.

Entomere. — In embryology *entomere* signifies one of the cells which go to make up the endoderm or hypoblast layer of the blastodermic vesicle.

Entomozoaria. *See* Nose, Foreign Bodies, Parasites (*Leeches, Centipedes, Earwigs, and Ascarides in the Nose*).

Entoparasite. — A parasite (intestinal worm) living in the interior of the body of its host.

Entophyte. —A plant parasitic in the body and causing a disease, such as Madura-foot or Mycetoma, and (perhaps) cancer.

Entozoa.—Animals living parasitically within other animals. *See* Parasites (*Ectozoa and Entozoa*).

Entropion.—An inversion of a cutaneous margin, such as the eyelid with the eyelashes, as opposed to *ectropion* (*q.v.*). *See* Conjunctiva, Diseases of (*Trachoma, Complications; Pemphigus, Sequel*); Eyelids, Affections of (*Defects in Position, Entropion*).

Enucleation.— The separation and removal of a tumour without much cutting (rather by shelling it out); also the excision of an organ, such as the eyeball. *See* Eyeball, Injuries of (*Penetration, Treatment*).

Enumeration.—In vital statistics *enumeration* refers to the decennial counting of the population. *See* Census; Vital Statistics (*Population*).

Enuresis. — Incontinence of urine, nocturnal or diurnal. *See* Urination, Disorders of (*Incontinence*); *see also* Belladonna; Hypnotism (*Treatment of Enuresis*).

Enzymes.

General

Special

See also Digestion and Metabolism; Immunity; Physiology, Protoplasm.

The terms organised ferments or fermenting organisms were formerly held essentially to denote the lower living fungi or bacteria, the unorganised ferments or enzymes being those substances secreted by animal and vegetable cells which can split up molecules by means which are not dependent on the life of the cells. It is probably advisable, however, now to discard our notions of the organised ferments, and simply to regard the ferments (the unorganised ferments of former writers) as identical with the enzymes. The splitting power of the organised ferments or micro-organisms is supposed to be essentially due to zymolysis, but such enzymes are certainly not excreted by all micro-organisms. In such cases the fermentative processes are probably carried on *inside the living cells*, *i.e.* that there are so-called active "Endoferments," which as a rule do not appear outside the cells, but which can be abstracted from them by means of certain processes involving the death of the cells. The ferment is often contained in the cells merely in the form of a zymogen, an antecedent body from which the true enzyme is formed under definite conditions only.

So far as can be determined from those pure and yet active preparations hitherto produced, the enzymes are proteids as regards their chemical nature. The reactions of such preparations, if used in not unduly dilute solutions, essentially correspond with those of the proteids.

Elementary analysis, too, supplies data which point to the fact that enzymes belong to the proteid group; for instance, pure pancreatic ferment (52·75% C., 7·51% H., 16·55% N., 23·19% O. and S., 1·77% ash). The actual position of enzymes in the proteid group is still the subject of discussion. Osborne, for example, regards diastase as a combination of albumin and proteose, while Wroblewski holds the same enzyme to be a substance closely related to albumose. Most of the enzymes can be precipitated by those substances commonly used to precipitate proteids (salts, *e.g.* ammonium sulphate, alcohol), and they are soluble in water and glycerine. Their activity is, however, impaired by all procedures which involve precipitation and purification. To obtain them in a pure form essentially the same processes are employed as in preparing proteids. From solutions containing proteid it is not only the enzymes which are precipitated, but ordinary proteid bodies as well, to which the enzymes adhere tenaciously. Enzymes are also carried down along with other inert deposits which may be produced in solutions containing enzymes. Thus Brücke employed precipitation by calcium phosphate for the production of pure pepsin, and precipitates of cholestearin also carry enzymes down with them. For further purification and separation, one can in the case of some enzymes make use of their peculiarity in not diffusing through membranes. On precipitation with alcohol, a more or less complete separation of enzymes from the proteids will be obtained by coagulating the latter with the alcohol; after long-continued action of alcohol and on subsequent treatment of the precipitate with water, the proteids are found to be almost insoluble, whilst the enzymes are still soluble in water.

In regard to their action the enzymes may be termed labile proteids, which apart from the cells are able in small quantities to induce chemical changes in other substances, whereby there arise bodies of lesser calorie. Such a change occurs without the enzyme itself forming any lasting chemical combination either with the substance which is being broken up or with the products of disintegration.

Thus in general there is no using up of the enzyme. Nevertheless a certain deterioration of power occurs in almost every solution containing enzymes, a dissociation of the enzyme, which is mainly to be referred to the influence of the temperature or to the products of disintegration which accumulate in the solution through the action of the enzyme.

The action of enzymes is in the majority of cases that of splitting up highly complex organic compounds into simpler substances, water usually entering the molecule in the process.

The so-called urea ferments, which convert urea into ammonium carbonate, furnish a typical example of this process—

$$CO{<}{}^{NH_2}_{NH_2} + 2H_2O \quad CO{<}{}^{ONH_4}_{ONH_4}$$

Such enzymes, therefore, may be said to act by hydration, a process which, as is known, may also be induced by simple chemical or thermic action. Thus fats, the higher carbohydrates, and true albumins, if they be heated with steam under pressure in an autoclave, may be converted into simpler compounds by hydration; the fats are broken up into glycerine and free fatty acids; starch is converted into grape sugar, albumins are broken up into albumoses, peptones, and amido-acids. In the case of many substances of this nature mere treatment with boiling water suffices to effect hydration, and especially if free acid or alkali be added. For example, the inversion of cane sugar, *i.e.* the splitting up into dextrose and levulose, is easily effected if the watery solution be boiled after the addition of an acid.

That the ferments act by hydration is proved not merely from the products of disintegration, but also from the interesting fact shown by Nasse that on the addition of diastase to a

solution of starch, the electrical conductivity of the fluid increases. From this one can deduce a formation by the ferment of ions, and consequently of dissociated molecules of water. But the specific nature of fermentation in contradistinction, for example, to the general hydrating effect of compressed steam, lies in the fact that the enzymes are able to split up only certain substrata. This fact has just lately been made the subject of more detailed investigation. E. Fischer, for example, has caused *emulsin* to act on the natural glucosides as well as on a number of artifical glucosides. Emulsin splits up those natural, and in part also those artificial, glucosides which belong to the β series, but not those of the a series. According to Fischer this elective power is to be explained by the asymmetrical construction of the enzyme molecule: if the action is to occur there must be between the enzyme and its substratum a definite relationship in the geometrical construction of the molecule.

"They must fit one another like lock and key."

This specific power of action manifests the close relationship of enzymes to protoplasm, for we know that cells in their action also show the same selective affinity for definite nutritive substances.

Almost all the substances which are capable of being split up by enzymes (German, "Fermentsubstrate") are of an esterlike nature, *e.g.* the fats. The proteids, too, seem to possess an esterlike grouping, at all events the ferments seem to act upon a similar esterlike group. It can also, in a certain sense, be termed a "saponifying" action. Nevertheless, it is difficult with our present knowledge to include all the actions of enzymes under the notion of hydration or saponification, and it is especially the so-called coagulating ferments which appear to prove obstacles in this direction. The action of rennin, for example, cannot be entirely explained by hydration. A process is accomplished by it which seems to be directly opposed to the other fermentations, in which, speaking generally, bodies which are but little soluble are transformed by enzymes into more soluble substances: what occurs here is that a substance in a state of solution, or at all events a *swollen condition*, becomes coagulated. Nevertheless one must bear in mind that rennin, at the same time (see below), separates a soluble compound from the casein, and that it is only the presence of lime which produces the coagulation, *i.e.* the separation of an insoluble compound of lime. Alexander Schmidt has already shown that in the coagulation of blood there occurs a synthesis of water with the proteids; and thus this process, which is seemingly at variance with the other fermentations, may also be ascribed to a process of hydration.

Lately, however, attention has been directed to a series of enzyme actions, which we have still greater difficulty in regarding as processes of hydration. We have here, however, to deal with processes which are not induced by pure ferments, but by vegetable and animal juices.

It may be assumed with certainty that we are here dealing with enzymes, even if, owing to their unstable condition, they are difficult to prepare in a pure form. We must assume that these enzymes are in still closer relationship to protoplasma than are those hitherto known. In this group there are the monosaccharide-splitting enzymes (zymase) and certain oxidations and reductions induced by vegetable and animal juices.

The effects of enzymes have also been termed katalytic, for they have been compared to certain chemical bodies, in the presence of which higher compounds become broken up into lower ones without the so-called katalytic substances suffering thereby any observable alteration. Thus nitrogen chloride is split up with explosion of its elements on contact with phosphorus and arsenic, which, therefore, in this case represent the katalytic substances. It is supposed that katalytic processes are concerned with the transmission of molecular movement, and Naegeli has similarly attempted to explain enzyme action. Opposed to this view is the fact that there is no hydration on the splitting up of nitrogen chloride, for example, and that in so far the simile does not correspond to most of the enzyme actions. Nor can the power which all ferments share in common, viz. of breaking up hydrogen peroxide into water and oxygen, be brought forward as a proof that zymolysis is a katalytic process.

The decomposition of hydrogen peroxide on contact with gold, silver, and manganese is certainly a typically katalytic process, but here again there is no hydration. Further, pancreatic juice, for instance, on being heated to 60° C., may be deprived of its power to split up hydrogen peroxide, whilst its diastatic activity is maintained.

A better analogy to enzyme action is found in the so-called *contact effect*, as is seen, for example, in the favourable influence which the presence of a trace of iodine exerts on the chlorination of toluol.

Since enzymes break up those bodies with large labile molecules into others with smaller and stable molecules, it follows that heat must be set free in every fermentative process. This elevation of temperature is, according to Duclaux, of essential importance. He compares the ferment to the match which sets a pile of wood on fire.

Ferment action is dependent on certain external factors. First, one should remember the sentence, "Corpora non agunt, nisi soluta." Then the enzymes are active only when in solution, but not when in a dry condition; they

are not destroyed by drying, but they require sufficient quantities of water for their action. If the solution contains, in addition to the enzymes, other substances in strongly concentrated form which have an affinity for water, the action of the enzymes will be thereby impeded, or possibly entirely prevented. Thus the presence in large amount of glycerine or of neutral salts acts as a hindrance. To the same cause, too, may in many cases be ascribed the fact that the enzymes have their activity reduced by the accumulation of their own metabolic products. Those forms of sugar which are produced by the inverting ferments, and peptone, which is formed by the proteolytic ferments, are bodies which have a strong affinity for water.

The temperature is another factor of great importance in zymolysis. The activity of solutions containing enzymes is, without exception, destroyed at the temperature of boiling water. This is another fact in favour of the proteid nature of enzymes. In this connection we have probably to deal with a coagulation of the enzymes. But the temperature which really "kills" enzymes is usually below 100° C., thus resembling that at which albuminous solutions coagulate. Destruction of almost all ferment solutions occurs on heating to 70°, but if other soluble bodies, *e.g.* sugar, be present, a higher temperature may sometimes be required. A great number of fermentations will even be destroyed if the solutions be kept at a temperature of 55° C. for half an hour. The temperature at which enzymes manifest the greatest activity (optimum temperature) is very variable. For most of the ferments of warm-blooded creatures it lies about 37° to 40°, especially for the peptonising enzyme. The diastatic ferments have generally their optimum temperature about 55°. It seems that the temperature necessary to "kill" depends on the optimum temperature, *i.e.* the higher the optimum temperature, the higher also the temperature required to "kill." Speaking generally, a gradually increasing destruction of the ferment commences whenever the optimum temperature is once exceeded. At − 10° C. the activity of most ferment solutions ceases, but is not altogether destroyed by such temperatures. Even the cooling of a solution down to − 100° C. does not destroy enzymes (Hahn). If the frozen solutions are again placed at a temperature of + 15° C. the enzyme action reappears. If in an absolutely dry condition ferments can be heated to very high temperatures (120°-160° C.) without showing any essential reduction in their efficacy when they are subsequently redissolved.

Proteids, as is well known, behave in a similar manner: they, too, suffer no loss in regard to solubility and power of coagulation from heating when in a dry condition.

While the fermenting organisms, excepting those which belong to the class of anaerobes, are for the most part unable to live in the entire absence of oxygen, and therefore incapable of setting up fermentation, the enzymes or unorganised ferments are independent of oxygen; they even act when in an atmosphere of pure hydrogen or of carbonic acid in the same manner as in the presence of air or pure oxygen. On the contrary, it seems that they are easily oxidised, and free access of air is consequently somewhat prejudicial to the efficacy.

A similar difference between the organised and unorganised ferments is manifested in their behaviour towards disinfectants. The enzymes are, in fact, more resistant than are the fermenting organisms. Æther, chloroform, benzol, toluol, and terpentine oil, in short all those antiseptics which act in the form of vapour, have almost no influence on enzymes. Sodium fluoride, hydrogen dioxide, and hydrocyanic acid, too, do not destroy enzymes. But, on the other hand, those antiseptics which coagulate albumin, such as corrosive sublimate, carbolic acid, and salicylic acid, and the fact points to the albuminous nature of the enzymes, have more influence on the unorganised ferments, but even then only if in greater concentration than would, for instance, be necessary to kill anthrax spores.

The behaviour of enzymes towards alkalies and acids is very variable. Whilst strong alkalies and acids destroy the activity of the enzymes, they often, if less concentrated, favour zymolysis.

It is more especially the activity of the hydrolytic ferments which is so dependent on the presence of weak alkalies or acids. The same phenomenon occurs in those hydrolytic processes which are effected without any fermentation. In such cases, *e.g.* the hydrolytic action of boiling water on starch, the process is materially aided by the addition of some acid. Fermentation is often intimately related to the presence of acids or alkalies. Pepsin, for example, acts only in an acid solution, trypsin only in an alkaline one, and thus the requisite reaction may be usefully employed in forming a classification of the ferments. The kind of acid used is of importance. The hydrochloric acid which is so useful for peptonisation cannot be displaced by an equivalent molecular quantity of acetic acid without a diminution of the fermentation resulting; hence one must in this case assume that the intermediate products which are formed, *e.g.* a combination of hydrochloric acid and albumin, are more susceptible to a further splitting up by means of the ferment.

I. Hydrolysing Enzymes

1. *Enzymes which split up Carbohydrates*

(*a*) *Diastatic Enzymes.*—By the term diastatic enzymes we mean those enzymes which are able to convert starch and allied bodies into ferment-

able sugar. These enzymes have a widespread distribution throughout the whole of the vegetable and animal kingdoms. They are of great importance for the nutrition of the vegetable and animal organisms, for they dissolve out from the reservoirs of reserve material the various forms of starch and convert them into the combustible forms of sugar.

The most important of the diastatic ferments is the *diastatic ferment of barley malt,* which is employed in brewing. Even while the barley is germinating, a portion of the starch is being converted into maltose and dextrin by the activity of the ferment. The ferment is not killed by the drying process, but it is only on the introduction of the ground malt into warm water that the diastatic action commences properly on the grain. The conversion of starch into dextrin continues until the ferment is killed on boiling the grain. Many people assume that barley really contains two enzymes, one which forms maltose from starch (maltase), and another which turns starch into dextrin (dextrinase). According to Beyerinck, dextrinase is not preformed in barley, but is artificially produced on drying the malt from an antecedent granulase, by this granulase losing its power of forming maltase, but retaining that of producing dextrin. The diastatic activity of malt is greatest at 54°-63° C. It rapidly diminishes on raising the temperature higher, and is entirely lost at a temperature above 75° C. Below 60° C. more maltose is formed, above 60° C. more dextrin. A faintly acid reaction of the fluid favours the activity of the malt diastase. Osborne and Wroblewski have lately succeeded in preparing diastase in a relatively pure condition. In particular it has been separated from a carbohydrate and its proteid nature confirmed. (According to W. it resembles an albumose.)

Closely resembling malt diastase, which was observed by Dubrunfaut in 1823, are other diastatic ferments which are widely distributed throughout the vegetable kingdom. They have been found in leaves, in seeds, and in the tubercles on the roots of many plants. Similar diastatic ferments are also formed and excreted by the lower fungi, and it is more especially the moulds (Aspergillus niger, Aspergillus oryzæ, Penicillium glaucum) which elaborate such ferments. The ferment of Aspergillus oryzæ is an article of commerce known as takadiastase, and it is also employed as a therapeutic agent. Further, not only the torulæ but also some forms of bacteria contain diastatic enzymes, *e.g.* bacillus orthobutylicus, granulobacter butylicum, and saccharobutylicum, the cholera, "potato," and lactic acid bacilli. Some of these diastatic ferments have been termed "glucases" because they convert starch and maltose into d-glucose. It is especially the diastatic ferment of maize malt, and of some torulæ which should be reckoned as glucases, because the diastatic action does not cease on the production of maltose, but continues, thus converting maltose into glucose. Green has shown that in the tubercles on the roots of some plants there exists a diastatic enzyme which converts inulin into levulose, and Bourgelot has proved that both Aspergillus niger and Penicillium glaucum contain "inulases."

Ferments which hydrolyse starch are also widely distributed throughout the *animal kingdom.* Let us, firstly, deal with the ptyalin of the saliva. The diastatic action of saliva was first observed by Leuchs in 1831.

It has been supposed to proceed from the microbes of the mouth, and not from a product of glandular secretion. Yet the great activity of saliva which contains no organic elements does not warrant that assumption On the other hand, it may be considered as definitely proved that ptyalin is not contained in the glandular cells as such, but as zymogen, from which latter substance ptyalin is only formed during the act of secretion. The saliva of the herbivora has the most powerful diastatic activity, that of the pure carnivora is inert. In the human subject the saliva, both from the parotid and from the submaxillary gland, has diastatic action, likewise the general saliva. According to Grützner ptyalin is not present in the submaxillary saliva of the pig or the rabbit. Ptyalin can be isolated in a relatively pure condition by the usual methods (precipitation by alcohol, extraction with glycerine, precipitation by calcium phosphate). The activity of ptyalin is greatest if the reaction be quite feebly alkaline, neutral, or just faintly acid. Hydrochloric acid is deleterious even in the strength of 0·075-0·1 per cent; similarly with other acids. But the simultaneous presence of albumin and peptones decreases the injurious action of the acid, hence there is a possibility, which cannot be excluded, that ptyalin, in certain circumstances, may even manifest its activity in the stomach. The optimum temperature for the action of ptyalin is from 38° to 41° (Paschutin). In a dilute solution its activity is destroyed at 65°-67° C. Ptyalin, after a short period of action on starch, forms maltose and dextrin; after a longer period dextrose is also formed from the maltose. Milk sugar undergoes no change by the saliva, but glycogen does. Boiled starch is much more readily converted into sugar than is unboiled starch.

The saliva of individuals who are suffering from mercurialism contains but little ptyalin (Claude Bernard).

Very closely related to ptyalin are other diastatic enzymes which have been found to possess an extensive distribution throughout the animal organisms. They have recently been more closely studied by E. Fischer and Niebel. In the blood serum of mammals, birds,

fishes, amphibia, and reptiles there is a diastatic ferment which converts starch, glycogen, and maltose into d-glucose. Milk sugar and cane sugar are not affected by this ferment. A diastatic enzyme could also be detected in watery extracts of the pancreatic juice, the thyroid gland, the testicles, and the mucous membrane of the stomach and small intestine.

In this instance, too, starch, glycogen, and maltose were hydrolysed. The diastatic ferment of the *pancreas* which we have now to consider is that which has been longest known, but as yet it has not been sufficiently separated from the other ferments of the pancreatic juice. The optimum temperature lies about 38°-41°, the temperature at which the enzyme is destroyed being 65°-70°. It is of practical importance to know that the enzyme is destroyed by the stronger acids and alkalies, that most of the salts, with the exception of Glauber's salt and sulphate of magnesia, tend to promote its action, and that quinine, morphine, and strychnine have no effect on it. The diastatic ferment of the pancreas is probably, like all the other pancreatic enzymes, contained in the gland merely in the form of a sparingly soluble zymogen, and that from this substance the enzyme is formed (in the presence of air, for example).

The *diastatic function* of the liver is also of great physiological significance. After what has been said in the introductory remarks with regard to the bacterial cell, it appears to be quite immaterial whether this diastatic function be due to the activity of the hepatic cells themselves or to an enzyme which has been separated from them. We have probably to deal here with an "endoferment," namely, an enzyme which during life is active inside the hepatic cells only, but which is set free after their death. The diastatic ferment of the liver is concerned with the conversion of glycogen into grape sugar; hence in the life of the animal it plays exactly the same parts as do the diastatic ferments in the vegetable kingdom in regard to the starches collected in the store-houses of reserve material.

(*b*) *Inverting Enzymes.*—The inverting ferments are those by means of which cane sugar and other disaccharides are split up.

The one best known is invertin (invertase), which converts cane sugar into d-glucose and d-fructose.

$$C_{12}H_{22}O_{11} + H_2O = \underset{\text{(d-glucose)}}{C_6H_{12}O_6} + \underset{\text{(d-fructose)}}{C_6H_{12}O_6}$$

Invertin was first discovered by Dubrunfaut in 1847 *in yeast*, watery extracts of which also contain invertin. Hence the enzyme is soluble in water and separable from the cells. On precipitation of a watery solution with alcohol the enzyme is carried down along with the albuminous bodies of the yeast; by extraction with water it is dissolved from the alcoholic precipitate, and separated from the albuminous bodies, which are now insoluble, and it can then, of course, be precipitated by alcohol from the watery solution. By fractional precipitation with ammonium sulphate (Wroblewski) a relatively pure preparation with 16·53 per cent N is also obtained, which from its reactions is an albuminous body, which, however, appears in nature in combination with a hitherto unknown carbohydrate. The optimum temperature lies somewhere about 56° C., while the temperature which kills the enzyme is about 70°. Acids in small amount ($\frac{1}{50}$ to 1 per cent acetic acid) are favourable to its action; acids in greater quantity, as well as quite a slight excess of alkali, tend to inhibit its action. Some of the salts (ammonium salts) aid, while others (potassium chloride) retard its activity, and alcohol is injurious. The quantity of inverted cane sugar is proportional to the amount of ferment present. Cane sugar up to 20 per cent favours the inversion. Yeast invertin dialyses and also passes through porcelain filters: it is therefore improbable that the inversion occurs in the substance of the cell only, as is assumed by many authors, and that such an excretion of invertin points to a degeneration of the yeast cells. At all events the elaboration and excretion of the ferment seem to be connected with the state of nutrition of the cells. Insufficient access of oxygen tends to promote the giving off of invertin. All yeasts do not produce invertin, yet the majority of the true yeasts and also the white and red yeasts appear to do so. Invertin seems to occur also in the leaves and flowers of the higher plants.

Inverting ferments have, moreover, been found in a few forms of fungi (Penicillium glaucum, Aspergillus niger), in B. megaterium, Proteus vulgaris, B. of Kiel harbour, B. fluorescens liquefaciens, etc.

In addition to the ferments which split up cane sugar there are in the vegetable kingdom, apart from the yeasts, other ferments which split up milk sugar into d-galactose and d-glucose, melitriose into d-fructose and melibiose, and melibiose into d-glucose and d-galactose.

In the *animal kingdom* the inverting ferments are likewise widely distributed. The splitting up of cane sugar in the intestinal canal of man, the dog, and the rabbit, must be referred to the action of invertin. Indeed, the extract of Brunner's and Lieberkühn's glands is inactive. There is, moreover, the possibility of the inverting ferment in the intestinal canal being identical with the diastatic enzyme.

Milk sugar is in general not decomposed in the small intestine of man and the animals; hydrolysis of milk sugar has only been proved to occur in the small intestine of newly-born children and of young calves, dogs, etc.

(*c*) *Enzymes which split up Glucosides.*—The

glucosides (esterlike combinations of monosaccharides, such as d-glucose or l-fructose, with bodies of a phenol or of an aldehyde nature) are broken up by a number of enzymes. According to E. Fischer the disaccharides which are split up by the inverting enzymes are to be considered as glucosides of sugar, hence it is not to be wondered at that other glucosides are also liable to be split up by ferments. Thus the inverting ferments of Penicillium glaucum and Aspergillus niger act upon amygdalin.

The best-known ferment of those which split up glucosides is *emulsin*, which was discovered by Liebig and Wöhler. By hydration it splits up amygdalin (obtained from bitter and sweet almonds by precipitation of a watery extract with alcohol) into sugar, hydrocyanic acid, and oil of bitter almonds.

There are other glucosides, such as salicin, chlorsalicin, helicin, arbutin, phloridzin, æsculin, daphnin, and coniferin, which are decomposed by emulsin. The optimum temperature appears to be from 20° to 30° C. Hydrochloric acid, even in a dilution of 0·135 per mille, abolishes its activity.

Another glucoside-splitting ferment, *myrosin*, occurs in watery extracts of white and black mustard seeds. It breaks up the myronate of potassium—myronic acid is a compound of a glucoside nature—contained in black mustard seeds into mustard oil, sugar, potassium sulphate, and some free sulphur.

2. *Enzymes which dissolve Albumin.*

These ferments have also a wide distribution throughout the vegetable and animal kingdoms, and are of great importance for metabolism. The true colloidal albumin is hydrolysed by the action of this enzyme, and then broken up into compounds which are capable of being absorbed and are fit for nourishment. Various observations point to the fact that in general almost all vegetable and animal cells are provided with some such enzymes as split up albumin. One important difference in regard to excretion must, however, be emphasised. There are but few forms of cells, in proportion to the total, which excrete these enzymes; many seem to give them off only if the death of the protoplasma occur in a certain sudden fashion. The majority of cells contain merely a zymogen which, in accordance with the constitution of the protoplasma, is converted inside the cell into enzyme. The subsequent splitting up of albuminous substances by this enzyme is usually an intra-cellular process too. In this fashion one can probably explain why it is that after animal organs have been minutely subdivided and digested with chloroform water peptic metabolic products have been seen to appear, whereas mere traces of these bodies exist in fresh organs, and especially in the liver (Salkowski). By pulverising and crushing the cellular substance of yeast a ferment of marked activity in splitting up albumin can be obtained, a ferment which is otherwise detectable only in old cultures by liquefaction of the gelatine (Hahn and Geret). A few other schizomycetes, such as typhoid and tubercle bacilli, have also, after similar treatment, yielded enzymes, the existence of which was formerly unknown. While there seem to be numerous intra-cellular enzymes or "endotrypsins" (Hahn, Geret) which split up albumin, the ferments as yet known which are secreted by cells, *i.e.* extra-cellular proteolytic ferments, are considerably fewer in number. On reflection the statement is explained by the fact that the proteolytic enzymes in contrast to the inverting ones, so far as is known, dialyse with difficulty or else not at all, and thus can only escape out through the cell wall under special circumstances.

The ferments which split up albumin may be regarded from two different standpoints:—

1. According to the form of digestive products which they produce.—Here again three types may be distinguished—(*a*) Ferments of the peptic type, which yield only peptone and albumose as products of digestion; (*b*) Ferments of the tryptic type, which produce albumose and peptone, but also amido acids and hexon bases as the products of splitting; (*c*) Those of the type of "yeast endotrypsin," which form merely traces of albumose, no peptone, but principally amido acids, and probably hexon bases. All of these decompositions can only be accomplished by the entrance of water into the albumin molecule.

2. The proteolytic ferments may also be classified according to the reaction which favours their working or which renders their action possible.—Thus we recognise—(*a*) Enzymes which split up albumin, but whose action occurs, or at any rate is aided, only if the reaction be acid, *e.g.* pepsin and yeast endotrypsin; (*b*) Those which require an alkaline reaction, *e.g.* trypsin and papayotin; (*c*) Those where the digestive process proceeds whether the reaction be faintly acid, alkaline, or neutral, for instance the enzyme in the juice of Ficus carica.

Pepsin.—For the formation, secretion, characteristics, mode of action, and digestive products of this enzyme see the article "Digestion." It is noteworthy that, while the optimum temperature for the pepsin of warm-blooded animals is 37°, that for the pepsin of cold-blooded animals seems to be 15°. Even though pepsin is destroyed to a slight extent during digestion, yet there is in practical circumstances very seldom any real deficiency of pepsin, but rather a want of acid. The presence of free hydrochloric acid is the fundamental factor in peptonisation, which can almost always be stimulated by the addition of fresh acid. Pepsin digests collagen, gelatine, and elastin, as well as the true albuminous bodies, while keratin, nuclein, and amyloid are not altered. Other ferments, such as emulsin

and malt diastase, are destroyed by a mixture of pepsin and hydrochloric acid. On the addition of 0·1-0·2 per cent hydrochloric acid boiled albumin is more easily digested than when not boiled. Pepsin may be prepared by the methods already mentioned for obtaining ferments; it can be got from the acid watery or glycerine extract of the gastric mucous membrane by precipitation with alcohol and calcium phosphate or with cholestearin. Peptic digestion is retarded or inhibited by salts, including metallic salts, if in concentrated form (*e.g.* 0·5-1 per cent sodium chloride), alcohol (20 per cent), salicylic acid (0·1 per cent), chloral, bromine, iodine, phenol, chloroform, large quantities of cane sugar (over 16 per cent), small quantities of æther, benzol, sulphurous acid, oil of bitter almonds, morphin, strychnin, digitalin, veratrin, and other alkaloids. Small quantities of glycerine and arsenious acid seem to exert no influence, but quinine in small quantities is said to hasten peptic digestion.

Trypsin.—The formation, secretion, characteristics, and mode of action of this ferment are described in the article "Digestion." The optimum temperature for the trypsin of warm-blooded creatures is from 37° to 40° C. Complete destruction of the ferment in watery solution occurs at about 70° C. The main factor for the powerful action of the ferment is an alkaline reaction. Soda seems to be most favourable in a strength of 0·2-0·5 per cent, but it may be increased up to 1·2 per cent. Acids, 0·1 per cent hydrochloric acid, for instance, usually abolish the activity of trypsin. Small amounts of salicylic acid (0·1 per cent) do not injure the enzyme. Large quantities of neutral salts retard its action, bile and the salts of the bile acids promote tryptic activity, while calomel does it little harm. In the preparation of trypsin, which, according to Kühne, consists of a coagulable albumin and peptone, while Löw holds it to be a body resembling peptone, the principle of precipitation by alcohol is as a rule employed. As the enzyme probably exists in the pancreas merely in the form of a zymogen, it is advisable first to leave the finely-minced organ lying for one day exposed to the air; the zymogen is then transformed into enzyme, probably by a post-mortem formation of acid.

Papaïn or *Papayotin.*—Papaïn occurs in the juice of Carica papaya, which has long been used by the inhabitants of India to soften meat. The leaves and the green fruits also contain the enzyme. Proteoses (deutero-proteose and true peptone) and also amido acids are formed as digestive products by papaïn. Whether the digestive products are identical with those formed by trypsin may be considered as extremely doubtful. Papaïn probably causes an actual process of splitting. Action occurs if the reaction be faintly acid, slightly alkaline, or neutral. The fermentative power increases up to 60° C., but active digestion proceeds even at 21° C. Hydrocyanic acid, boric acid, and phenol do not prevent the action of papaïn. The enzyme has been prepared from the juice by precipitation with alcohol, etc. From its reaction it, too, seems to be a proteid. It is highly probable that some of the commercial invalid food preparations which have been brought forward as peptones, albumoses, etc., are produced by papaïn.

There are other tryptic enzymes in the vegetable kingdom which are closely related to papaïn. A peptonising enzyme, which with 0·2 per cent hydrochloric acid has a powerful digestive action, bas been obtained from the juice of Ficus carica, as well as from dried figs. The germinating seeds of many plants (Green) contain albumin-splitting enzymes, which, however, do not appear to be excreted, but which are to be regarded as endoferments in the above-mentioned sense of the word. They are of great importance for the development of the plants, act in acid solution, and form albumoses, peptone, leucin, and tyrosin. Similar to them are the proteolytic enzymes of the schizomycetes (Penicillium) and of the insect-eating plants (*e.g.* the enzyme of Nepenthes (Vines), which forms leucin and tyrosin in an acid solution. In this group, too, must be placed the endotrypsin of saccharomyces, obtained from bruising and crushing the yeast cells, also appearing in old cultures, which is distinguished from all other proteolytic ferments by the fact that peptone and albumoses can hardly be detected among the decomposition products, whilst leucin and tyrosin appear in great abundance. This endotrypsin acts in acid solution.

The proteolytic enzymes of bacteria, which are apparent in cultures by the liquefaction of gelatine, are distinguished from most of the other enzymes found in the vegetable kingdom by being efficacious only in alkaline solution. They are rapidly destroyed by weak acids, particularly the inorganic ones, and likewise by the heating of a watery solution over 70°. Gelatine, especially when in liquid form, is more readily acted on by the bacterial enzymes than are the true and solid proteids. Sunlight reduces the power of this ferment, but carbolic acid and sublimate in weak solutions do not do so. The *elaboration of ferment* continues only as long as the vegetative growth of the bacteria lasts. The *excretion of the ferment* is not, however, dependent on the growth of the bacteria. On the contrary, the view that ferment escapes in abundance from old, dead, or degenerated cells must be regarded as correct. Fermi, among others, has shown that all those bacterial forms which liquefy gelatine contain such peptonising ferments—thus, cholera vibrio, bacillus prodigiosus, pyocyaneus, the pyogenic staphylococci, and anthrax bacilli. These bacterial forms

secrete the enzyme even during the life of the cells. But there are also other bacteria which do not liquefy gelatine, yet which contain similar enzymes, which can be obtained from the bodies of the bacteria by pulverisation and crushing (typhoid and tubercle bacilli, Geret and Hahn). Hence there are endotrypsines here too, as well as in saccharomyces. These proteolytic bacterial enzymes have doubtless a definite relationship to the formation of toxine, and hence to the production of disease; but hitherto so little light has been thrown on this relationship that further details thereof cannot at present lay claim to any importance.

II. Enzymes which coagulate Albumin

The best known of these ferments is rennin (chymosin, cheese ferment). This is more especially found in the calf's stomach, but is also found in the stomach of young mammals in general, existing as zymogen, which is easily converted into the true rennin by acidulation. It also occurs in the gastric juice of older mammals (including man), birds, and amphibia. The action of rennin consists in coagulating the casein of milk. More accurate investigations have demonstrated that soluble calcium salts must always be present if this process is to occur, and that, on the other hand, the presence of acids is by no means requisite as was formerly thought. The precipitation of milk by acids is a process quite distinct from its coagulation by rennin. In the latter process (according to Hammarsten) the soluble compound of caseinogen and lime contained in the milk is first split up into a soluble compound of casein and lime and into whey-albumin which resembles albumose. If there are, however, soluble lime salts present, the soluble compound of casein and lime unites with them to form double salts which are insoluble in fluids whose reaction approaches the neutral point. As milk fulfils both these requisite conditions (soluble lime salts and a neutral or amphoteric reaction) casein is consequently separated as a firm insoluble coagulum. The removal of the soluble lime salts from the milk by dialysis or by the addition of ammonium oxalate, abolishes the power of rennin, therefore, in the same manner as does a high degree of alkalinity of the milk. Feeble acidulation, on the contrary (introduction of carbonic acid), is somewhat favourable, for more lime salts are thereby rendered soluble; prolonged boiling and sterilisation, and also the diluting of milk, tend to inhibit coagulation by rennin, because the relative amount of insoluble lime salt is thereby diminished. For the production of powerful extracts dried calves' stomachs are extracted with dilute acids, water solutions of common salt, or glycerine. Glycerine extracts retain their power for long periods. The optimum temperature for calf's rennet is the same as that of the body, but in the cheese-making industry lower temperatures are employed (20°-25° C.) The temperature at which rennin is killed is about 66°. The time required for coagulation is dependent on the amount of rennin present.

Milk which has been in contact with rennet at the temperature of the body for only a few seconds, is immediately coagulated by boiling, evidently because boiling favours the formation of the insoluble compounds of casein and lime. If, however, milk and rennet remain in contact for a longer time at the body temperature, it is considerably longer before the milk is coagulated. The action of rennin is, however, generally speaking, one of the most rapid forms of enzyme action.

The succus entericus, pancreatic juice, and urine, are also said to contain a milk-curdling ferment.

These ferments have likewise been observed in the vegetable kingdom, but their physiological significance is not clearly understood. Thus a milk-curdling ferment is contained in the sap of Ananassa sativa, of Carica papaya, and of the fig-tree, in the flowers too of many of the Compositæ (artichokes, Carina acaulis), and also in Galium verum, and in some kinds of thistles. These vegetable milk-curdling ferments have also been employed in cheese-making, and especially so in southern countries.

Fibrin Ferment.—This ferment, which produces the coagulation of blood, seems to be widely distributed among the bodily cells. Extracts from red and white blood corpuscles, and from animal and vegetable tissues, cause coagulation of the plasma of horse's blood, which is obtained by cooling down to 0° and filtering, and which does not coagulate spontaneously. These extracts likewise cause coagulation of blood in the living animal body after intravenous injection. The coagulation of blood which has left the arteries always seems, under the usual natural conditions, to occur by the breaking down of white blood corpuscles, and the leucocytes are accordingly regarded as being the main seat of the zymogen which is converted into fibrin ferment on the destruction of the blood corpuscles. Fibrin ferment is prepared by exposing defibrinated blood or serum to strong alcohol for some months, and the coagulum is subsequently dried in the air and extracted with a little water for half an hour. It is probably of the nature of a globulin which loses its enzymatic action by being heated to 75°. The coagulation of blood is, according to recent investigation, analogous to but not identical with the coagulation of milk. Coagulation of the blood takes place by means of an enzyme-fibrin ferment, which transforms soluble fibrinogen into insoluble fibrin. The importance of soluble lime salts in this action has been recognised for some time by scientists; but only

recently has it been definitely settled that these salts act upon the forerunner of fibrin ferment, transforming it into the active enzyme. They play no part in the transformation of fibrinogen into fibrin, the latter containing no more calcium than the former. The nuclei of the leucocytes, according to Lilienfeld, contain a substance termed neucleohiston, which may be split up into two components, one of which, termed leuconuclein, aids coagulation, while the other is called histon and tends to inhibit coagulation. Nucleic acid, which is a non-albuminous body, is also said to cause coagulation. All processes which retard the breaking down of blood corpuscles also inhibit the coagulation of blood. This occurs, for instance, if the blood be cooled down, if it be passed into salt solutions or into vessels smeared with fat, whereby contact of the blood corpuscles with the wall of the vessel is avoided. Coagulation is also prevented by the addition to the blood of such substances as diminish the amount of soluble calcium salts, *e.g.* solutions of oxalates. The excretion of fibrin is, on the other hand, hastened by stirring the blood, because the destruction of the corpuscles is thereby aided.

III. Ferments which split up Fats

Claude Bernard was the first to prove that there is an enzyme in the pancreatic juice which splits up fats into free fatty acids and glycerine. This process is of physiological importance, because the fatty acids which are set free unite with the alkali of the succus entericus to form soaps. Any neutral fat still present becomes emulsified by these soaps, *i.e.* broken up into minute globules, and thus rendered suitable for absorption through the intestinal wall. This fat-splitting enzyme acts in neutral, faintly acid or alkaline solutions, but greater alkalinity is very favourable to its activity. The activity of the enzyme increases up to 60°, but is destroyed by boiling. The ferment is also found in the so-called "dried pancreas of Kühne," and can therefore be precipitated by alcohol.

Henriot has lately found in the serum of various species of animals, and of the horse in particular, a fat-splitting enzyme. Only in non-essential points does this enzyme differ from the steapsin of the pancreas, and the physiological action of both seems to be identical (saponifi ation).

Furthermore, fat-splitting enzymes were detected by Green and others in certain vegetable seeds, especially if they were germinating, for instance in fruit of Papaver somniferum and Cannabis sativa, in flax seeds, and in maize. Similar enzymes have been found in Penicillium glaucum and Aspergillus niger. They have not as yet been detected in bacteria, but nevertheless the rancidness of fats is probably due to the action of some such fat-splitting enzymes formed by bacteria.

IV. Urinary Enzymes

Formerly it was generally supposed that the micrococcus ureæ alone was able to convert urea into ammonium carbonate. Miquel has lately shown that 60 different bacteria (cocci, bacilli, and sarcinæ) which were cultivated from the air, river water, and drainage, possess this power, and he has proved that their action is due to enzymes which may be separated from them. If the cultures be passed through porcelain filters in the absence of oxygen—for the enzyme is very easily oxidised—fluids are obtained which are almost completely sterile, yet which decompose urea. The optimum temperature of urase is 55°. The gradual accumulation of ammonium carbonate, occurring through the activity of the urase, is injurious both to the growth of the bacteria and to the enzymes produced by them.

V. Monosaccharide-splitting Enzymes

1. *Zymase.*—E. Buchner, aided by M. Hahn, was the first to prove that the fermentative action of torulæ may proceed without living cells being present, and consequently that the action is in all probability due to an enzyme formed by these cells. The proof is afforded by the following process which has been elaborated by M. Hahn. One kilogram of freshly-crushed yeast is ground up with one kilogram of quartz sand and 200 grammes of silica, and the moist mass is subjected to hydraulic pressure of 300 to 500 atmospheres. In this way one obtains an albuminous fluid which passes through filters of paper or silica, which contains only a few torulæ, but which nevertheless produces marked fermentation. Since the juice of the yeast or yeast plasmin also contains invertin, it is not only grape sugar which is quantitatively converted into alcohol and carbonic acid, but also cane sugar, levulose, milk sugar, and less markedly d-galactose and glycogen. Lactose and l-arabinose are not fermented. The addition of antiseptics (*e.g.* toluol or potassium arsenite) does not injure the fermentation. Zymases can be precipitated by alcohol with consequent loss of fermentative power. In a dry condition it withstands heating to 100°. It retains its fermentative power for months if dried *in vacuo* at 30°. The optimum temperature lies between 30° and 37°, and the fermentative activity of zymases ceases almost entirely at about 55°.

We are still uncertain as to whether zymase is excreted by the living cells or active merely as an endoferment inside the cells. At all events torulæ, which are first carefully and completely dried and then heated to 120°-140° so that they are killed, produce fermentation, and therefore contain zymase.

2. *The Glycolytic Blood Ferment.*—Fresh defibrinated blood, especially that of the dog, is able to a limited extent to decompose grape sugar into carbonic acid and water. The activity

of the blood is very quickly diminished on standing at the room-temperature, so that it is sometimes completely abolished a few hours after it has left the arteries, but if kept in an ice cupboard it is somewhat better maintained. The optimum temperature is from 37° to 38°. The activity is destroyed on heating the blood to 55°. As the blood serum is inert the fermentative activity must be connected with the blood corpuscles, especially the red ones. Lépine supposes that it is a ferment formed by the pancreas, and that the accumulation of sugar in the blood and urine of diabetic persons is due to the deficiency of this ferment. The glycolytic power of the blood is very sensitive to all interference, to the addition of antiseptics, etc., and consequently many difficulties are thrown in our way in studying those questions which deal with the origin and importance of this fermentation. This action of the blood might also be regarded merely as an oxidation process. (See below.)

VI. Oxidising and Reducing Enzymes

Very manifold oxidation processes have been observed to occur in the blood and in animal organs, *e.g.* in a broth made from fresh liver which has been washed free of blood, and these processes have been regarded by some authors as due to enzyme action. In such broths, for instance, benzylalcohol is converted into benzoic acid, salicylaldehyde into salicylic acid, certain chromogens, such as paraphenylendiamin, into other colours, and hydrogen peroxide is decomposed. The glycolytic action of the blood may also be regarded as an oxidation. At present we are in doubt as to whether, in these instances, we are really dealing with an oxidising ferment. *The reducing action*, too, which animal organs have been observed to exert on methylene- and alizarin-blue cannot be definitely classified as a fermentation. On the other hand, the fact ascertained by Rey-Pailhade that an extract of torulæ produces sulphuretted hydrogen from flowers of sulphur—an action also possessed by the cell-free juice of torulæ—is to be regarded more as the action of a reducing enzyme. This enzyme is termed "phylothion."

There is no doubt that certain of the bacterial poisons—tetanus toxin, for instance, which even in extremely small quantities acts with great power in the animal body—are closely related to the ferments. The action, too, of antitoxin, and of agglutinin, recalls that of the enzymes. The power which the blood naturally possesses of destroying bacteria and globulins is still more closely related to the fermentations. The normal fresh blood and blood serum of animals and human beings has, as is well known, the power to kill bacteria in considerable numbers, and similarly to dissolve the blood corpuscles belonging to a different species of animal. Both these capabilities of the normal blood and blood serum are abolished on heating to 55°. The simplest explanation of these processes would be offered by the assumption of a labile proteolytic enzyme occurring normally in the blood.

These points will be more fully considered in the article "Immunity."

Eosin.—A coloured compound produced by treating resorcinol fluorescein with bromine; chemically it is tetra-brom-fluorescein ($C_{20}H_8Br_4O_5$); it is a valuable cytoplasmic stain, much used in microscopy, especially in combination with hæmatoxylin (which stains the nucleus).

Eosinophile.—The property of being easily stained by acid dyes, possessed by the granules of certain leucocytes and myelocytes. These granules are of large size, contrasting with the fine granules of the neutrophile cells. *See* Blood, Physiology and Clinical Investigation of.

Eosinophilia.—An increase in the percentage number of the eosinophile leucocytes in the circulating blood. It is present in leukæmia, in paroxysmal asthma, in various skin diseases, in gout, in scarlatina, and in parasitic diseases, especially ankylostomiasis, trichinosis, and filariasis. It is of considerable importance in the diagnosis of many of these diseases. In suppurative conditions the presence of eosinophile cells in the blood is of favourable prognostic import. An ordinary polymorphonuclear leucocytosis is usually associated with a decrease or even entire absence of the eosinophiles. *See* Dermatitis Herpetiformis (*Pathology*); Leucocytosis (*Eosinophile*); Pemphigus (*Pathology*); Parasites (*Trichina Spiralis*).

Epactal Bone.—The interparietal or epactal bone is a small accessory bony nucleus occasionally found in the sagittal suture near its posterior end; it is also called the os Incæ (because it is supposed to be commoner in Peruvian skulls).

Epanalepsis.—Recovery from sickness; analepsis.

Eparsalgia.—A morbid state due to violent exertion and overstrain, such as cardiac dilatation, enteroptosis, hæmoptysis, and hernia.

Ependyma.—The neuroglial lining of the cerebral ventricles and of the central canal of the spinal cord; it is subjacent to the epithelium; inflammation of this layer is called *ependymitis*.

Ephelis.—Freckles or lentigo. *See* Skin, Pigmentary Affections of (*Classification, Sexual*).

Ephemeral Fever. *See also* Tropics, Unclassed Fevers (*Continued Fevers of Short Duration*).—*Definition.*—The term *ephemeral*

fever is commonly applied to any febrile state not dependent on infection or local affection, and of shorter duration than twenty-four hours. If such a febrile state continues for a longer period, say for three or four days, the term febricula is often used, but no real distinction can be drawn between ephemeral fever and febricula.

Nature.—The term ephemeral fever is usually made to cover a considerable variety of conditions which differ greatly from each other in their nature and origin, and which do not all strictly conform to the definition given above. The most important of these are the following:—

(1) *Abortive Infections.*—During epidemics of any of the ordinary infectious diseases one meets with cases of persons attacked with malaise and often sharp fever, which subsides in the course of a day or two, often by crisis, without the development of any rash or any other local symptoms. Persons exposed to the infection of a disease from which they have already suffered may be attacked in this way. The diagnosis in such cases is often extremely uncertain, and recovery is so rapid that it is often a very difficult question to decide whether or not the patient can be allowed to resume his occupation without running the risk of carrying infection.

(2) *Fever following Chill.*—"Catching cold" is sometimes attended by a very considerable febrile reaction in the entire absence of any local affection, or with a sore throat or nasal catarrh much too mild to account for the amount of fever. As the fever is attended by a considerable degree of malaise, with loss of appetite and coating of the tongue, it is often set down, without any sufficient reason, as due to gastric catarrh.

(3) *Fever with Dyspepsia.*—Dyspeptic attacks, especially in children, may be attended by considerable fever. Fever may be due in some instances to the absorption of toxic substances either present in the food when eaten or arising in the course of digestion.

(4) Fever may result from prolonged and violent *muscular exertion.* Such fever may be associated with great tenderness of the muscles and petechial hæmorrhages in the skin over the muscles affected.

(5) Fever following *exposure to the sun.*—Even in this country prolonged exposure to a hot sun may be followed by some febrile reaction. This may be associated with erythema or even some blistering of the parts of skin exposed to the sun.

(6) Fever may arise as the result of breathing *foul odours* or gases from badly-ventilated sewers. Such fever may come on very rapidly, with nausea and vomiting, and may in some cases be followed by collapse or coma. The air in well-ventilated sewers appears to be innocuous.

(7) *Tropical Ephemeral Fever.*—New-comers to tropical climates are liable to be attacked by an ephemeral fever characterised by sudden onset, with considerable rise of temperature, malaise, dryness of the skin, and all the ordinary accompaniments of fever, lasting from twenty-four to seventy-two hours, and passing off frequently by crisis with copious sweating. This form of fever, occurring during the process of acclimatisation, is probably produced by the altered conditions of life, especially by defective metabolism with retention of effete products, rather than the result of any specific infection.

A similar fever may attack old residents and natives, and is then frequently complicated with malaria.

(8) Fever due to unrecognised lesions. In young children and lunatics especially it is often extremely difficult to make certain whether any lesion is present which would account for fever.

Cases of fever of short duration may also be met with which cannot be brought under any of the above headings. Such cases have been described as occurring especially in elderly women, in women at the period of the menopause, and in the subjects of enlarged prostate. Similar cases may be met with in otherwise perfectly healthy adults. Probably most cases of ephemeral fever must be regarded as belonging to one of the groups above mentioned rather than as examples of a specific disease of no known etiology, without symptoms except those of fever, and with no known pathology.

Symptoms.—The symptoms usually set in abruptly, sometimes with some preliminary indisposition. The temperature usually ranges from 101° to 103°, but not infrequently it rises higher. There is usually loss of appetite, the tongue is coated, and there may be uneasiness in the epigastrium. The skin is hot and dry. The urine is scanty and high-coloured. There may be slight delirium at night, especially in the case of children. Herpes on the lips is a common symptom, especially in cases following chill. Such cases are sometimes spoken of as herpetic fever, though the herpes is obviously only a symptom, and one which may not appear till the fever has abated. The fever usually terminates by crisis, which may be accompanied by copious sweating.

The diagnosis depends on the absence of any local cause of fever, of symptoms of any of the specific fevers, and especially of the characteristic eruptions, and upon the rapid subsidence of the fever.

Prognosis.—The prognosis is entirely favourable in healthy subjects. It must be somewhat more guarded in the case of debilitated patients, and especially of alcoholics. In alcoholic subjects especially danger may arise from visceral complications.

Treatment.—The patient should be kept at rest in bed, clad lightly but sufficiently, and

protected from draughts. The room may be darkened. The patient should be kept as quiet as possible.

A milk diet is suitable in most cases. Nourishment should be administered at short intervals in small quantities at a time. If thirst is present, fluids (*e.g.* barley water) may be allowed freely. A laxative should be ordered at the onset, and a diaphoretic mixture during the course of the fever. Tepid sponging of the face and the extremities, or sponging with vinegar and water, or eau de Cologne and water, will often diminish restlessness and favour sleep.

When the fever subsides, the patient should not be allowed to resume his occupation too soon. In most cases tonics are desirable during convalescence.

Ephialtes.—Nightmare (Gr. ἐπί, upon, and ἅλλομαι, I leap).

Ephidrosis.—Sweating or any disease accompanied by sweating.

Epiagnathus.—The monstrosity in which there is absence of the upper jaw (*Taruffi*); it differs in nature from *epignathus* (*q.v.*).

Epiblast.—The outer layer of the blastoderm. *See* EMBRYOLOGY; FŒTUS AND OVUM, DEVELOPMENT OF.

Epicanthis or **Epicanthus.**—A malformation in which a fold of skin stretches across the inner end of the interpalpebral fissure and obscures the inner canthus. *See* EYELIDS, AFFECTIONS OF (*Congenital Defects*); STRABISMUS (*Apparent, Epicanthus*).

Epicarin.—A product of the action of beta-naphthol on creasotic acid, used (as solution or ointment) in psoriasis, eczema, and scabies.

Epicomus.—A form of double monster, in which an accessory head is attached to the top of the head of the autosite (*St. Hilaire*).

Epicondylalgia.—Pain in the region of the external condyle of the humerus, probably situated in the muscles and tendons.

Epicranium.—The soft parts covering the cranium; the scalp.

Epidaurus. *See* BALNEOLOGY (*Greece*).

Epidemic.—Diseases which come to one part of the world from another and usually distant part, and which affect a great many people at the same time, are usually said to be *epidemic*. Instances are enumerated in the article on EPIDEMIOLOGY (*q.v.*). *See also* DROPSY, EPIDEMIC; DYSENTERY (*Epidemic Gangrenous Rectitis*); HAEMOGLOBINURIA; HERPES ("*epidemic*"); INSANITY, ETIOLOGY OF (*Epidemicity*); MENINGITIS, EPIDEMIC CEREBRO-SPINAL; MUMPS OR EPIDEMIC PAROTITIS; STOMATITIS (*Aphthous*).

Epidemiology.

INTRODUCTORY

Definition, Endemicity, Sporadicity, Specificity, Receptivity

THE term *epidemic* was originally applied to a disease attacking a number of persons at the same time or in close succession. Thus, Paulus Ægineta says, "We call those diseases epidemic and common that attack many persons together," and the word is often used in this sense at the present day. Haeser in his *History of Epidemic Diseases* treats of ergotism and scurvy as epidemic maladies. Ergotism, as we know, is an intoxication, and scurvy a disease of malnutrition; yet, as both frequently affect large numbers at the same time, they may properly enough be classed as diseases, ἐπί, upon, δῆμος, the people. Hecker, in the same way, includes the dancing mania of the Middle Ages along with the Black Death among epidemic diseases, because, although psychical in its nature, it spread by a sort of moral contagion and became widely prevalent. In common life we speak of an epidemy of suicide, of sunstroke, of lead colic, and so forth, meaning no more than that these diseases are unusually common at a given time, and without regard to their nature or causation.

Although this use of the word is etymologically correct and sanctioned by the best authorities, we shall, in this article, make a distinction between common and epidemic diseases—restricting the term epidemic to that group of infective or micro-parasitic maladies which has the common property of spreading from time to time in a community. This property of attacking larger or smaller numbers of a population simultaneously or in succession implies a common origin of the units constituting an epidemy. The individual cases must be connected either by filiation, the one from the other, or by derivation from a common source of infection.

This definition excludes intoxications, dietic and psychical diseases, as well as those arising from physical agencies, such as heat or cold.

The distinction formerly recognised between pestilential and epidemic diseases, founded on theoretical and obsolete views of their causation, has now little more than historical interest. Some modern authorities, however, reserve the name of pestilences to plague, yellow fever, and cholera, on account of their extension, from time to time, over large regions and the terrible mortality to which they give rise when they become widely epidemic. Epidemiologically these three diseases present certain notable peculiarities. They are each endemic in one or more centres from which epidemic extensions take their start. They also exhibit in a high degree those variations in spreading and killing power which, in a more or less marked way, characterise all epidemic maladies. These peculiarities, and others that could be mentioned, do not, however, require that plague, cholera, and yellow fever should be placed in a separate class by themselves.

The relation between epidemic and endemic diseases requires a few words of explanation. The fact that certain diseases are restricted to, or specially prevalent in, particular localities must have been a matter of common observation from the earliest times. The treatise of Hippocrates "On Airs, Waters, and Places" is, in fact, a dissertation on what we should now call endemic influences. A formal distinction, however, between endemic and epidemic diseases is not to be found in the works of the Greek, Roman, or Arabian physicians, but appears to have been made for the first time by Galeazzo di Santa Sofia in his *Liber de Febribus*, published in 1514.

Strictly speaking, there is no natural class of endemic maladies. Diseases, the most diverse in their characters, have their favourite or exclusive haunts. Endemicity depends on one or other of the following circumstances:—(*a*) The telluric or climatic conditions peculiar to a region favour the saprophytic growth of a pathogenic microbe, as in the case of cholera, which finds in Lower Bengal and some other places the conditions which enable it to maintain a continuous existence. (*b*) Conditions peculiar to a particular region or locality favour the life of some insect or other animal which serves as the intermediate host of a pathogenic parasite, as in the instance of the mosquito in relation to malaria, or which acts as the carrier of a disease, as in the case of the tsetse fly. (*c*) The habits of life and social circumstances of a people in some cases account for a disease clinging to a locality. Overcrowding, want of ventilation, the housing of cattle along with man, and imperfect burial of the dead, are common to all plague centres. (*d*) The presence or absence of other forms of animal or vegetable life may determine the endemicity of a disease. Moulds, for example, favour the growth of the yellow fever bacillus.

The division of diseases into epidemic and endemic, as if they formed two mutually exclusive classes, comprehending between them all infective maladies, is quite inadmissible The same disease may be at once endemic and epidemic. Cholera, as we have just said, is endemic in Lower Bengal, but at intervals it becomes so prevalent there as to assume the character of an epidemy, and from this endemic centre it spreads over continents. Malaria is endemic in more or less defined regions, but in certain seasons it becomes epidemic in its endemic haunts, and extends far beyond its usual limits. The converse of epidemic is not endemic but sporadic (σποράδικος, scattered).

An epidemic disease often occurs sporadically in inter-epidemic periods. This results from an attenuation of the virus which permits its infecting only a few specially susceptible individuals, from a want of facilities for diffusion, or from a temporary insusceptibility on the part of the population. Whether an infective disease be sporadic or epidemic will depend on two factors: (*a*) the resistance of the virus, that is, its capacity of retaining its vitality outside the body; (*b*) the facilities which the contagium has for effecting an entrance into susceptible subjects. It is the latter factor that is of the greatest importance in this connection. Gonorrhœa is an infective disease, the virus of which is reproduced in great abundance and for a considerable time; but the gonococcus rapidly loses its infective properties outside the body, and the infection being only communicable by the direct application of the virus to certain mucous surfaces, which in ordinary conditions are not exposed to the contagion, it is impossible that the disease should occur otherwise than sporadically. The virus of measles, on the other hand, although by no means resistant, being readily communicable by simple proximity to the sick, has the opportunity of becoming rapidly diffused through a community.

It follows that if a sporadic infective disease should undergo a change in its mode of communication it may assume epidemic characters. Such a change took place in the case of syphilis in the end of the fifteenth century. The contagium, from being localised and communicable by sexual intercourse only, became generalised in multiform eruptions on the skin and mucous membranes, known as the "pockes," by which it was communicated in the ordinary intercourse of social and family life, and hence became rapidly diffused over Europe. As soon as the disease resumed its old type, it lost its epidemic characters. The difference between a sporadic and epidemic disease is, thus, not in the nature of the virus, but in the mode in which it is propagated.

We have omitted from our definition of an

epidemic disease the property of specificity, which is, in a sense, implied in the fact that it is caused by a *contagium vivum.* If we use the term, the meaning we put into it must be wide enough to cover the following facts:—(*a*) Microbes presenting marked morphological and biological differences give rise to the same disease. The vibrios of cholera differ so much from one another that they are supposed by some to belong to different species.

(*b*) A complex of microbes may be concerned in producing the lesions of a disease. In most cases one microbe can be clearly identified as specific; the others, so to speak, form its retinue. The presence of the specific microbe determines the appearance of the others. It has been proved that the products of the cultivation of the bacillus of yellow fever promote the growth of the coli bacillus, the staphylococcus aureus, the proteus vulgaris, and the streptococci. This explains the frequent presence of these organisms in yellow fever, in many cases of which the specific bacillus is crowded out by these intruders. In non-amœbic dysentery a variety of organisms are always present, and it is impossible at present to say if any one of them, to the exclusion of the others, is concerned in originating the dysenteric process. Whether this form of dysentery is strictly specific, in the sense of being always due to one organism, is uncertain, but that it often assumes epidemic characters is abundantly evident.

(*c*) A great variety of symptoms and lesions may be produced by the specific microbe itself apart from the existence of a mixed infection, such as we meet with in yellow fever. Simple variation in virulence does not, as a rule, materially affect the nature of the lesions, but the port of entrance of a microbe has a remarkable influence on the gravity of the symptoms, and even on the lesions of a disease. We know how the manifestations of plague differ according as infection takes place by the skin, the respiratory passages, or the alimentary canal. Inoculated small-pox is a mild disease compared with that contracted in the ordinary way. If we were to go out of the field of strictly epidemic diseases, we should find a remarkable illustration of the influence of the port of entrance on the character of a disease by comparing the symptoms and lesions of cutaneous and pulmonary anthrax. To sum up, we may say that, with a few doubtful exceptions, all epidemic diseases are specific, and the symptoms and lesions produced by the pathogenic organism are the same under the same conditions. The marked differences in these respects observed in certain diseases depend either on the virus effecting its entrance by different ports, or on the association of other organisms causing a mixed infection.

The presence of the specific organism of an epidemic disease is not sufficient to determine an epidemy. For this, among other things, a susceptibility or receptivity on the part of a community is essential. Susceptibility to all infections is increased by conditions which lower the resistance of the system generally, or that of the surface through which infection takes place. Insufficient nourishment, muscular exhaustion, mental fatigue, exposure to cold or excessive heat, and vicissitudes of temperature all predispose the body to infection, although one set of conditions does not predispose to all diseases indifferently. The experiments which establish the influence of these agents in rendering the body amenable to particular contagia are too well known to require that they should be mentioned here. But the vast importance of predisposition as determining whether infection shall, or shall not, follow exposure to the specific cause is not sufficiently recognised. A few illustrations of the effect of predisposition in determining the epidemic spread of disease must suffice.

The germ or germs of dysentery are ubiquitous. It is sufficient to subject a body of men for a time to exhaustion, want, alternations of heat and cold to ensure an outbreak of dysentery, and once produced, the disease becomes epidemic, and spreads to those not subjected to these hardships. The history of every long and trying campaign furnishes evidence of the influence of predisposition in giving rise to epidemic dysentery.

The microbe of typhus is unknown, but it, too, appears to be widely diffused and ready to come into evidence as soon as circumstances affecting the susceptibility of a community favour its pathogenic activity. It is sure to make its appearance sooner or later among those whose resistance has been broken down by want or disease, if they are confined in dark, filthy, unventilated dwellings on sea or on land. Creighton records a very remarkable instance of typhus being generated (if we may use the expression) in an Egyptian frigate, having on board 476 men, 200 of whom were convicts. The voyage to Liverpool was long and stormy, preventing ventilation 'tween decks. The vessel, besides, was in a horrible state of filth. There was much sickness of a diarrhœal kind among the men, but *no fever* appeared on board. On arriving in the Mersey these men, who were themselves free from fever, communicated typhus to the English who were brought into contact with them, or who went on board the vessel. In all thirty took the infection, and eight died.

Kelsch has recently shown how certainly the fatigues consequent on the annual manœuvres in France, by their effect in reducing the resistance of the body to the virus, are followed by outbreaks of enteric fever. In the same way malarial fever readily becomes epidemic among those subjected to the hardships of war, or who suffer from want. It is for this reason notably a disease of the poor. Duboué's experience in France is that of all who have seen much of the

disease in the tropics. "L'infection palustre," he says, "est rare, très rare dans la classe aisée. Les huit dixièmes des cas d'impaludisme que j'ai observés, je les ai vus dans la classe peu aisée ou misérable, et les quelques exemples que j'ai notés parmi les gens riches s'expliquent presque toujours par des imprudences hygiéniques." We shall have occasion in the sequel to notice the effect of an increased vulnerability of the air-passages produced by atmospheric conditions in determining the seasonal incidence of small-pox and measles.

What we call immunity is a lessened susceptibility, original or acquired. This plays an important part in limiting the spread of epidemic diseases. What would have been the fate of our race if one attack of an epidemic disease afforded no immunity from another? When a cholera wave passes over India the population acquires a partial immunity, lasting, as a rule, for two or three years. Hence epidemic waves follow with considerable regularity every fourth year. It has been shown that the ingestion of the cholera bacillus in food or water, while it may fail to give rise to the infection, nevertheless confers a certain degree of immunity. Abel and Claussen found the vibrio in the stools of thirteen out of seventeen persons who had been brought into close contact with cholera patients. They had imbibed the virus without succumbing to the disease, and had doubtless in this way acquired a partial and temporary immunity. We may thus suppose that a very large number of a community among which cholera has been raging, and who have escaped an attack, become temporarily immune. Otherwise it would be difficult to understand why the disease should not spread every year in India, seeing that in the worst outbreaks those who have acquired an immunity by passing through an attack form only a small proportion of the population. A similar process of temporary immunisation by imbibing non-infective doses of the virus takes place in other infections. Is it not possible, then, that something similar occurs even in the case of contagious diseases? The increase of virulence observed in the earlier stages of an outbreak of measles or small-pox is generally explained on the assumption that at the beginning of an epidemy only the more susceptible are attacked, in passing through whom the virus gains in intensity. This theory, whether true or not, implies that others who imbibe the virus do not contract the disease on account of their greater resistance. We may suppose that in the case of those who receive the virus without developing the disease, the pathogenic organism is destroyed *in situ* by the action of the cells; but it does not follow that the bacterial products are without effect on the economy. This view of the possible immunising influence of non-infective doses of the virus of contagious diseases is not to be dismissed as purely fanciful, for Copeman has shown that protection against the effects of subsequent vaccination or inoculation is afforded by the introduction of the virus, although there may be no local manifestations. Should it be proved that the action of the virus—say of small-pox or measles—in doses insufficient to give rise to disease confers a temporary immunity on those subjected to it, some difficulties in connection with the decline of epidemics and the intervals between successive outbreaks would be removed or lessened.

Methods of Study.—Classification.—Epidemiology is a branch of natural history—that branch of it which treats of the micro-organisms to which the microbes of epidemic diseases belong. A complete natural history of these organisms would include, among other things, their epidemiology. The life of an important group of these parasites is limited to man, and their life-history is comprised in their epidemic activity. The most interesting phase in the life of the others is not that which is passed by them as harmless saprophytes, but that in which as parasites they take their share in the tragedy of human life, bring misery into the domestic circle, and, as in the case of plague, determine the fate of empires and modify the progress of civilisation.

The natural history of the parasites of epidemic diseases embraces their bacteriology and their epidemic manifestations. Bacteriologically, we have to study them in their relation to other organisms to which they are allied or which affect their growth; their morphology; the media in which those of them that can be grown outside the body can be best cultivated; the influence of physical agencies—temperature, humidity, and so forth—on their growth and virulence. But this is only a fragment of their natural history. We have to observe their behaviour as human parasites in order to discover how they spread, the influence of meteorological agents on their diffusion, the vehicles by which they are introduced into the body, the personal and social conditions which influence infection, the circumstances that determine the decline and extinction of epidemics, and much more that cannot be learned by bacteriological research. But our knowledge of the natural history of these organisms is not complete until we know something of their epidemic history—their varying prevalence and fatality at different epochs, and the symptoms they have exhibited in past times.

We must beware of concluding too positively from the behaviour of an organism under experimental conditions how it will comport itself in its epidemic career. Cholera cultivations die out rapidly when placed in the soil, but an outbreak of cholera occurred in 1890 at Puebla de Rugat in Spain, which could only be accounted for on the supposition that the vibrio had retained its vitality in the soil for a period of five years. Under the direction of Pettenkoffer 5 litres of a bouillon containing an estimated

number of seventy-two millions of the typhoid bacillus were poured into a well containing 680 litres of water. They had all disappeared by the third day. Our experience of actual epidemics of water-borne typhoid is very different from the results of this experiment. We must be equally on our guard not to infer from what we see of an epidemic disease at the present day that it has always presented the same characters. In recent outbreaks of plague little has been heard of carbuncles, petechiæ, and other eruptions. Yet so characteristic were carbuncles of this disease, as seen in Europe in the sixteenth and seventeenth centuries, that it was often spoken of as *carbuncular* plague, and the eruptions known as *the tokens* were looked upon as more certain evidence that a disease was plague than the buboes themselves.

The micro-organisms of some of the more important diseases are still unknown, but those that are known belong to the coccus, rod, or screw forms of the schizomycetes, or to the hæmatozoa.

Classifications based on their morphology and modes of reproduction, however necessary for the bacteriologist, are of little service to the epidemiologist, for they fail to bring together groups of disease having common epidemiological features. The symptoms, lesions, and epidemiological characters of a disease afford no certain indication of the bacteriological class in which we are to look for its cause.

A classification based on the parasitic habits of the micro-organisms and the modes in which they are communicable is that which is most useful to the epidemiologist.

One important class of epidemic diseases consists of those caused by *obligatory* parasites. These organisms do not grow outside the human body. External conditions—light, air, temperature, humidity—are relevant to their prevalence only in so far as they (*a*) attenuate their virulence or destroy their vitality; (*b*) favour or hinder their diffusion; (*c*) increase or diminish the susceptibility of the body generally, or that of the surfaces through which infection takes place, to their invasion.

To a second class belong diseases caused by *non-obligatory* parasites capable of saprophytic life outside the body. This class is usually divided into two groups: diseases due (*a*) to facultative saprophytes, or organisms essentially parasitic, but capable of growing more or less vigorously in external media; (*b*) to facultative parasites, or organisms essentially saprophytic in their habits, but capable of invading the human body. The distinction is a real one as applied to non-epidemic infections. The bacillus of tetanus, for example, is a truly facultative parasite. Its life is that of a soil saprophyte, but when accidentally introduced into the body it is capable of growing in the tissues. The distinction, however, between facultative saprophytes and facultative parasites is more arbitrary in the case of the organisms of epidemic diseases. The most saprophytic of them develop a high degree of parasitism when they are epidemic. It is doubtful, indeed, if any saprophyte can give rise to an epidemy until its virulence has become exalted by successive transmissions through the human body. In other words, facultative parasites must become, in a sense, facultative saprophytes before they can spread extensively. The cholera vibrio, for example, is looked upon as the type of a facultative parasite, but it loses its virulence by continued growth as a saprophyte. This is true at least in non-endemic regions, and it is open to doubt whether the cholera germ, even in places where it is endemic, can maintain indefinitely the virulence necessary to give it spreading power, unless by frequent passages through the human body.

Some of the non-obligatory parasites approach closely in their epidemic characters to those of the obligatory group. The scarlet fever germ, for example, whatsoever it may be, is capable of saprophytic growth, as milk epidemics prove, but when widely epidemic, scarlet fever appears to spread almost exclusively by contagion, and its epidemic characters are then practically those of a disease due to an obligatory parasite.

The same may be said of diphtheria. Its bacillus grows as a saprophyte, and during its epidemic prevalence the saprophytically grown virus often gives rise to the disease, but there is reason to believe that when the disease is epidemic it is mainly spread by contagion. When not grown from time to time on the body it loses its virulence. It will thus be seen that epidemicity implies an increase in the parasitism of microbes, those most akin to saprophytes in their habits approximating to the characters of facultative saprophytes, the latter, again, to those of obligatory parasites. It seems highly probable, indeed, that all infective microbes were originally saprophytes, and it cannot be confidently affirmed that any one of the obligatory group has so completely lost its ancestral habits that it cannot grow in some medium outside the body.

There is some reason for believing that the bacillus cultivated by Copeman and Klein from variolous crusts by incubation in a hen's egg is the long-sought-for virus of variola. In that case, the most obligatory of all parasites will have been proved to be capable of saprophytic life; but from all we know of small-pox we may rest assured that in respect to the actual mode of its propagation it is strictly obligatory. When small-pox or measles are introduced into an island with a limited population, and not in frequent communication with places where these diseases are always more or less in evidence, they die out as soon as the susceptible are exhausted. Neither of them can maintain itself in the surroundings of man until a sufficient

number of unprotected persons accumulate to set agoing a new outbreak.

It follows from what we have said that a definitive classification is at present impossible. The following groups, however, indicate to some extent the epidemic affinities of the more important of the diseases with which we are concerned:—

1. Diseases caused by obligatory parasites and spread by contagion. To this class belong small-pox, measles, whooping-cough, mumps, chicken pox, and less certainly typhus and cerebro-spinal fever.

2. Diseases caused by non-obligatory parasites which spread by contagion; but as their contagia are capable of saprophytic growth, they are also diffused by infection. In this group of contagious-infectious diseases are scarlet fever, diphtheria, and erysipelas, and probably relapsing fever, influenza, and dengue.

3. Diseases caused by non-obligatory parasites which are spread mainly by infection. The microbes of this group, although derived more or less remotely from a previous case, multiply outside the body, and are introduced into the system by means of air, water, food, or in the instance of plague, and perhaps of yellow fever, by inoculation. This group comprises cholera, plague, Malta fever, enteric fever, dysentery, and yellow fever.

4. Malarial fever is caused by a hæmatozoon. It is communicable by the sting of a malariated mosquito, but the etiology of the disease points to other modes of infection. It is still uncertain if the parasite of blackwater fever is distinct from that of malaria.

Isolation of, and avoidance of communication with, the sick, or contact with contaminated articles are the means of limiting the spread of diseases belonging to the first class. Defective sanitation is only of importance in promoting their spread in so far as it predisposes the body to infection.

Isolation is still the most important means of limiting the spread of the second group. But here the need of protecting milk and food from contamination is obvious. Sanitation also comes into greater prominence, inasmuch as an impure soil may serve as a breeding-place for the germs, while noxious effluvia and sewage gases may at once predispose to attack, and serve as vehicles for the infection.

In respect to the third class the first indication is to prevent the introduction of the germ into a healthy country or locality by measures of quarantine, inspection, and disinfection proper to the particular disease to be guarded against, and suited to the circumstances of the community. Of primary importance for the safety of a community is attention to purity of air, soil, and water, with free ventilation and sunlight, so as to deprive the microbe of breeding-places, prevent the contamination of the vehicles by which the contagium is conveyed into the system, and subject the virus to the action of the natural agencies that are antagonistic to its vitality.

The prevention of malarial diseases requires that a country be rendered unfit for the life of its insect host by means of drainage and cultivation. Individual prophylaxis does not come within the scope of this article.

To sum up: for an epidemy of a disease belonging to the first and second groups there must be (*a*) the virus; (*b*) a susceptible population; (*c*) free inter-communication between the sick and the susceptible. For the epidemic prevalence of a disease belonging to the third group there must be (*a*) the virus; (*b*) a breeding-place for it outside man; (*c*) means of transport from place to place; (*d*) a vehicle for its diffusion; (*e*) a susceptible population. Isolated cases of an epidemic disease may occur when some of these conditions are absent, but in order that an infectious disease should become widely diffused, they must all be present.

General Epidemiology

Epidemic Movements—Law of Anticipation—Associations and Antagonisms—Evolution and Involution of Epidemics

Epidemic Movements.—The distinguishing feature of epidemic diseases, as the name implies, is their alternating periods of quiescence and recrudescence, but other movements, less obvious, because more gradual in their evolution, have also to be considered. Some of the more important epidemic phenomena fall under the following heads:—1. Secular mutations occurring during the course of centuries. 2. Multiannual nutations, to use the phrase of Ransome, or fluctuations in prevalence and virulence extending over periods of from ten to fifty years. 3. Epidemic waves or explosions recurring at more or less regular intervals of a few years. 4. Seasonal fluctuations. 5. Oscillations at irregular intervals measured by days or weeks.

I. Secular Mutations.—Under the term secular mutations are comprised (*a*) changes in the relative importance of a particular disease or class of diseases, developing during the course of ages; (*b*) the appearance of new epidemic diseases, or the extension of old ones to regions from which they had been previously absent; (*c*) the extinction or modification of epidemic diseases; (*d*) the temporary assumption of epidemic characters by sporadic infectious maladies.

(*a*) A study of the history of epidemic diseases shows a certain malady or class of maladies coming to the front on the epidemic scene, playing the chief rôle for a longer or shorter time, and then retiring to give place to others. We have an example of this kind of secular movement in the predominating importance of plague

in the sixth and seventh centuries, and, again, from the fourteenth to the middle of the seventeenth century. Other instances are afforded by the remarkable prevalence of typhus in the seventeenth and eighteenth centuries, its gradual decline in recent years, and the varying prevalence of malaria, dysentery, and the contagious class of diseases in different historical periods.

(*b*) The English sweating sickness is the best-known example of the advent of a disease of which no trace is previously to be met with in history. Its sudden appearance in 1486, its repeated periods of apparently complete extinction, followed by new outbreaks, and its final disappearance in 1551, form an altogether unique episode in the annals of epidemiology. Whether dengue is to be reckoned among new diseases is not so certain, but its history does not reach back beyond the last quarter of the eighteenth century. Cerebro-spinal meningitis, if not new, escaped recognition up to the year 1837.

The epidemic diseases mentioned by Hippocrates are malarial fevers of the various types now known, continued fevers of long duration, presenting some of the features of Malta fever, possibly diphtheria, dysentery, summer cholera, mumps, erysipelas, puerperal fever. Plague, too, was known in his day, although it is not described in any of his authentic works. The list of epidemic diseases has increased considerably since the time of Hippocrates by the evolution of new diseases or by the extension to Europe of maladies previously restricted to other regions. Respecting the existence of small-pox and measles in ancient times Dr. Adams says: "After having read, we may say, every word of every ancient writer on medicine that has come down to us, we can confidently affirm that the Greeks and Romans are altogether silent on the subject (of small-pox and measles), and we are indebted to the Arabians for the earliest accounts we have of these diseases." In this conclusion we agree, and we are even inclined to doubt the vast antiquity claimed for small-pox in India and China. The historic evidence of this antiquity is doubtful; the improbabilities of its being restricted for ages to one country, great. Had small-pox prevailed in the East from remote antiquity, as some hold, it is difficult to understand why it should not have rapidly spread to Europe if the disease had then possessed the contagious character it now exhibits. The constant intercourse between the East and West in ancient times afforded ample opportunities for its spread.

But whatever may be the antiquity of small-pox and measles, their extension to Europe, which is one of the most notable secular movements of epidemic diseases, does not date before the sixth century. In recent times other diseases have extended their limits. Yellow fever has in our day become acclimatised in Brazil, and in the past and present centuries it has frequently overrun great parts of North America, and has made repeated incursions into Southern Europe. One of the most remarkable instances of the extension of a disease previously confined to a comparatively limited area is that afforded by the repeated pandemics of cholera during the present century.

(*c*) History, too, affords examples of the extinction or modification of old diseases. No form of sickness now known corresponds to the plague of Athens as described by Thucydides, to that of Antoninus (166-68 A.D.) described by Galen, or to that of Cyprian in the third century. The modifications in prevalence and virulence which epidemic diseases have undergone in historical times are apparent. Malarial fever and dysentery dominated the pathology of England in the days of Sydenham; they are at present not only among the rarest of diseases, but they have lost much of their old virulence. When do we hear now of the pernicious attacks described by Morton which "sub larva algoris funesti, vomitionis indesinentis, choleræ morbi, colicæ ventriculi, apoplexiæ, syncopes, spasmi universalis, pleuritidis, vel alterius morbi secure delitescens, medicum a scopo suo non raro abducit"? Typhus fever, so prevalent in past centuries, seems to be verging towards extinction. On the other hand, the quasi-epidemic diseases of the respiratory organs—pneumonia and bronchitis—have gained in importance, and since the first appearance of Asiatic cholera the mortality from diarrhœal diseases has enormously increased, although of late years it seems again to be somewhat on the wane. The mortality from this class of diseases per million living in London since the beginning of registration has been as follows:—

1838-40	1841-50	1851-60	1861-70	1871-80	1881-90
274	782	1030	1040	949	748

This increase has not been confined to London or England. According to Lombard the deaths in Berlin from diarrhœa and cholorine formed 18·6 per 1000 of the deaths from all causes from 1835 to 1838; by 1868-69 they had risen to 109; in 1872 to 134; and in 1873 to 173 per 1000. Is this great increase caused by the acclimatisation in Europe of an attenuated descendant of the vibrio of Asiatic cholera?

(*d*) The temporary assumption of epidemic characters by sporadic infective diseases is best exemplified in the epidemic spread of syphilis in the end of the fifteenth century. Something analogous, but not so much to the point, is observed in the spread of leprosy in Europe in the Middle Ages, and in New Caledonia and the Sandwich Islands of late years.

Space forbids us entering into a discussion of the causes of these secular movements. They

may be referred to one or more of the following circumstances:—(*a*) Variations in the prevalence and virulence of the *contagium*, brought about by altered conditions under which the micro-organism is grown, or the association of pathogenic microbes with non-pathogenic organisms which exalt or attenuate their virulence. (*b*) Changes in the circumstances and habits of peoples as regards food, dwellings, occupations, modes of life, and whatever affects the susceptibility to disease. (*c*) Changes in the facilities for the spread of certain diseases by social and political upheavals and movements of population. (*d*) The assumption of parasitic characters by microbes formerly purely saprophytic.

II. Multiannual Fluctuations or Nutations.—In following the course of a contagious disease, such as small-pox, measles, whooping-cough, scarlet fever, or diphtheria during a long series of years in a community in which it is constantly more or less present, it will be remarked that it has periods of slowly increasing and decreasing mortality, quite distinct from the epidemic explosions recurring at shorter intervals. These long-period fluctuations appear on a chart as alternate ebbs and flows, or swells and depressions, on which the short-period outbursts appear as waves. Or rather a diagrammatic representation of the course of one of these diseases through a series of years presents something of the appearance of an undulating mountain range, studded with numerous abruptly projecting peaks, arising alike from height and hollow—those shooting up from the depressions being often the highest. These undulations or long-period swells are frequently spoken of as epidemic cycles, but if carefully examined they are found not to be strictly cyclical, as they are neither of equal height nor do they recur at regular intervals. In diagram 1, Chart I., showing the ratio of deaths from small-pox per 1000 of the deaths from all causes in London from 1700 to 1800, we observe a distinct increase in the mortality during the first decennium, the swell attaining a maximum from 1715 to 1720. It then subsides slowly and irregularly to 1733. Then follows an ebb up to 1745, when another long swell begins, which seems to end about 1783. Another upward movement is in progress when the epidemic force of the disease was arrested by the introduction of vaccination. In diagram 3, Chart I., representing small-pox in Sweden from 1749 to 1800, it will be seen that there is a marked swell from 1749 to 1773, corresponding with that observed in the London chart for the same period. Unfortunately, we do not have the material for deciding how far these swells coincide in different countries, but the curves of small-pox in London and Sweden in the second half of the eighteenth century suggest that these swells are not entirely local. In diagrams 1 and 2, Chart II., similar swells and ebbs are to be observed in the mortality from measles. The first swell in the London chart seems to have been about its height in 1838. There is a distinct fall from 1846 to 1852, when the ebb is at its lowest. So far as can be gathered from the statistics of the pre-registration period, the increase began in 1834. It thus covered a period of about 18 years. Another more indefinite swell extends from 1853 to somewhere about 1871, forming another period of 18 years. Then follows a steady rise from 1872, which is beginning to subside when the record closes. The very marked rise in the measles mortality in New York (Chart II., diagram 2) coincides with the latter part of the London period. Whooping-cough (diagram 3, Chart II.) shows a very slight rise from 1841 to 1850; it then remains pretty much at the same level from 1851 to 1870, after which the fall has been steadily progressive. We are not, however, to conclude that this is a permanent downward movement, for an examination of the charts for Sweden, published by Ransome (*Epidem. Soc. Trans.* vol. i. N.S.), shows that this disease has what he calls a nutation period of about 50 years.

Scarlet fever and diphtheria are eminently mobile diseases. They vary much more in prevalence, and especially in virulence, from time to time than do diseases of the purely contagious class. The scarlet fever seen by Sydenham was so mild that when it proved fatal "the sick died of his doctor." Graves observes that scarlet fever assumed a very benign type in Dublin soon after the year 1804, and continued mild up to 1831. "It then increased in severity, and in 1834 the disease assumed the form of a destructive epidemic." There would thus seem to have been a period of three or four years during which the pathogenic agent of the disease was slowly gaining in virulence before the epidemy attained its maximum intensity. The fluctuations in the mortality from this disease from 1859 to 1892 are depicted in diagram 4, Chart II. During the twenty years 1871-90 scarlet fever has been gradually losing ground in London and elsewhere, but those acquainted with its history will hesitate in ascribing this decrease, as is often done, exclusively or mainly, to the influence of isolation hospitals. Diphtheria presents the same variations in prevalence and virulence which we observe in scarlet fever. Its virulence may, indeed, be often observed to increase during the progress of an outbreak, the early cases presenting the characters of a simple sore throat, developing later into the most malignant type of the disease. From this mobility of type, diphtheria appears for a series of years in widespread and fatal epidemics, then subsides, to recur in local outbreaks only at irregular intervals. We have no exact statistics to guide us, but according to Hirsch there was

a general remission of the disease, except in France, from the end of the eighteenth century until about the year 1858. From this date it has been one of the most fatal epidemic maladies of young persons not only in Europe, but in Asia Minor, the United States, Canada, and other countries.

During its long periods of activity diphtheria undergoes marked fluctuations in prevalence. The first swell in London covers the period 1859-72. A second and greater swell succeeded, which, so far as diagram 2, Chart I., shows, reached its height in 1889. These alternate ebbs and flows in the diphtheria death-rate of London do not correspond to those observed in New York and Philadelphia.

The causes of these swells are obscure. We are unable to say with any certainty whether

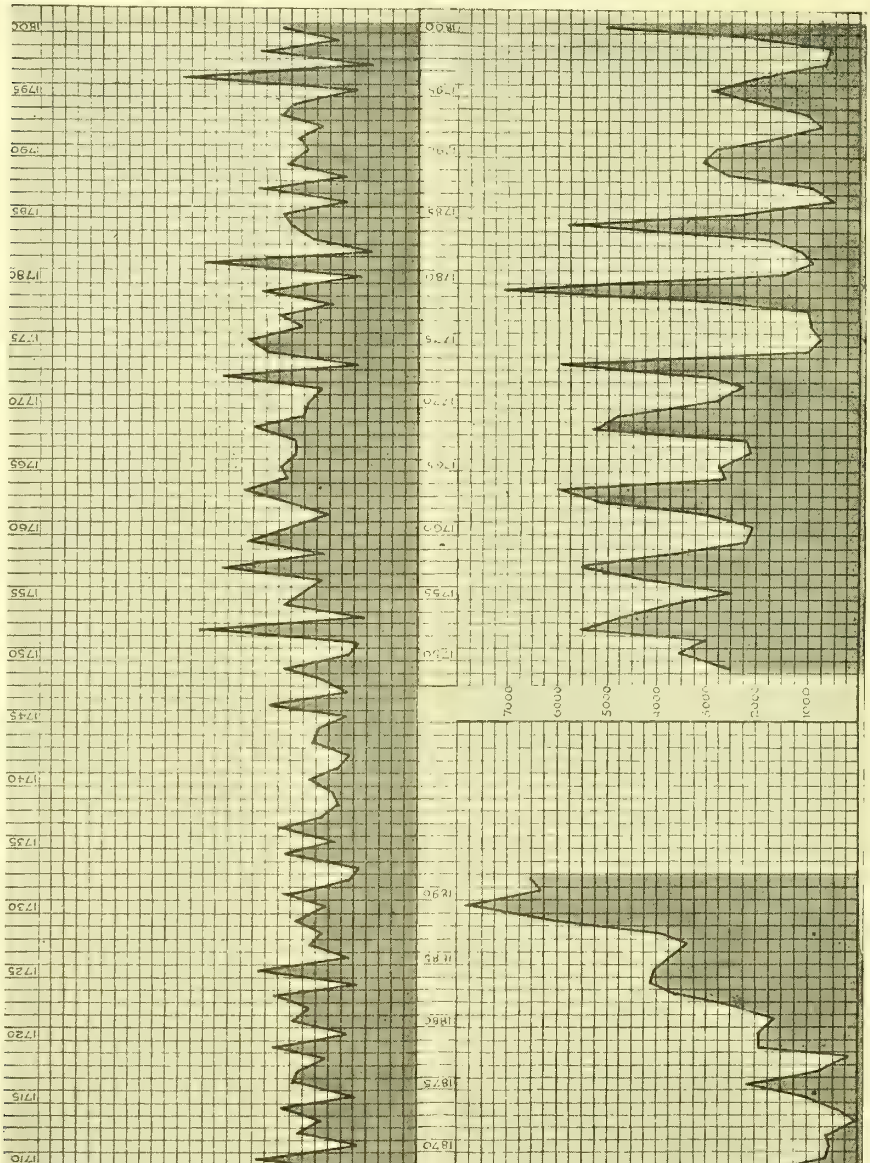


CHART I.

Diagram 1.—Small-pox in London, 1700 to 1800. Rates per 1000 of deaths from all causes.

Diagram 2.—Diphtheria, mortality per million. London, 1859-1891.

Diagram 3.—Small-pox. Sweden, 1749-1800.

as respects small-pox, measles, and whooping-cough they denote an increased prevalence only or an increased virulence as well. The long-period movements of scarlet fever are certainly dependent upon, or associated with, an increased virulence of its contagium, although how this is brought about is unknown.

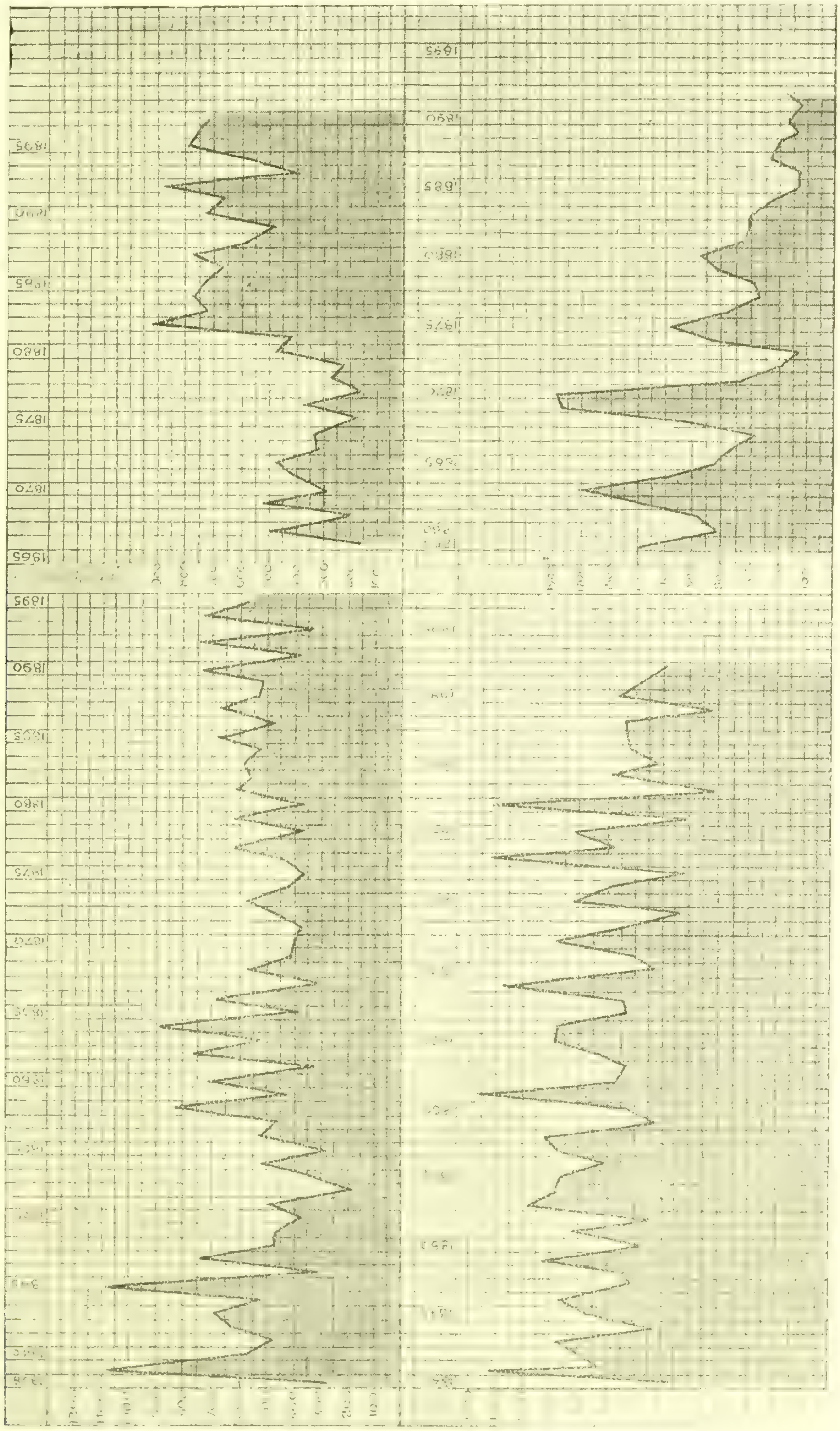


Diagram 2.—Measles, actual number of deaths. New York, 1865-1896.

Diagram 1.[1]—Measles, annual mortality per million. London, 1838-1895.

Diagram 4.—Scarlet Fever, mortality per million. London, 1859-1892.

Diagram 3.—Whooping-Cough, mortality per million. London, 1840-1892.

CHART II.

[1] In diagram 1, Chart II., the figures at margin have been placed one line too high. To obtain the correct ratio of deaths per million add 50 to numbers indicated on diagram.

The greatly diminished small-pox mortality consequent on the introduction of vaccination was followed by a notable increase in the deaths

from measles and whooping-cough. The possibility, therefore, of these long waves being to some extent complementary to other diseases affecting infant life is not to be overlooked. It will be observed from the charts that as the death-rate from whooping-cough in London has fallen, so has that from measles risen. Perhaps the diminishing mortality from scarlet fever from 1870 may also have contributed to increase the fatality of measles during the same period. But after a careful examination of the deaths from infantile diseases in New York, we fail to find a decrease in any of them to which the enormous increase of measles from 1880 onwards can be ascribed. As one cannot die of two diseases, the suppression or decline in one cause of death will leave a greater number of possible victims for another. But this being admitted, it really explains nothing, for if the increase of measles be the result of the diminished ravages of scarlet fever and whooping-cough, to what, we ask, is the decreasing fatality of these diseases due?

Some have sought for an explanation of such phenomena in atmospheric constitutions favouring the prevalence of this or that disease or group of diseases. It is here that a few words may be said about the doctrine of constitutions, which occupies such an important place in the history of epidemiology.

The constitution or *katastasis* of Hippocrates was an annual one, determined by sensible states of the weather—heat, cold, dryness, humidity, the direction and force of the winds, and so forth. Diseases, according to him, were not caused by the weather prevailing just at the time when they appeared, but by this in relation to the weather in the seasons preceding. As Bacon puts it, an epidemic constitution "results from a precedent sequence and series of the seasons of the year." This doctrine was founded not on conjecture but on observation, and will remain true, for a certain class of diseases and within certain limits, for all time. It largely explains the varying prevalence of the diseases with which Hippocrates was acquainted in different years and seasons.

Sydenham's constitution differed from that of Hippocrates. It was not an annual one, but of uncertain duration, and it was not caused by sensible but by occult qualities of the atmosphere, and finally it was conceived in order to explain phenomena which, to a large extent, were outside the observation of Hippocrates—such as plague, spotted fevers, and small-pox. Epidemic diseases, to use Sydenham's own words, "are engendered through occult and inexplicable changes in the atmosphere, and continue their devastation during the persistence of the mysterious skiey influences in question." There is nothing in nature corresponding to this conception.

It is clear that neither the constitution of Hippocrates nor that of Sydenham throws any light on these multiannual fluctuations with which we are dealing. Another doctrine is that of Haeser, who asserts, on historical grounds, the existence of two, to some extent mutually exclusive, constitutions, which he calls respectively a typhous constitution, during which typhus and typhoid fevers, plague, and malarial fevers predominate; the other an exanthematous constitution characterised by a general and excessive prevalence of scarlet fever, small-pox, measles, dysentery, diphtheria, and puerperal fever, all of which, he thinks, are related maladies. These constitutions are supposed to alternate at varying periods, extending over, it may be, twenty or thirty years, and determining the type of disease over continents. He supposes that these constitutions are the result of meteorological and cosmic influences affecting alike the vegetable and animal kingdoms. The historic evidence in support of these views does not appear conclusive. The facts that have just come under our notice indicate that the periods of prevalence of the individual members of the exanthematic group do not always correspond. Their ebbs and swells do not coincide as they should do if Haeser's doctrine were true. If these movements are caused by atmospheric and cosmic influences, we should have to postulate a separate constitution for each member of the exanthematous group. It must be admitted that these nutations are amongst the most inscrutable of epidemic phenomena.

III. Short-period Waves.—It will be seen from an examination of Chart II. that epidemic waves of measles follow one another with considerable regularity every other year in London and New York. The same holds true of most large cities. This biennial wave is scarcely less marked in England and Wales as a whole, although the years in which the wave occurs in the country do not always coincide with those in which it appears in the capital. In sparsely populated localities these waves occur at longer and quite irregular intervals. In the seventeenth and eighteenth centuries the intervals between the successive waves of measles in London were less regular than at the present day, but the tendency to biennial explosions was even then quite apparent.

Chart I. shows that there was generally an epidemic outburst of small-pox every other year in London in the eighteenth century. The interval between the waves is four years in Sweden (*see* Chart I.), instead of one year as in London.

The intervals between the successive explosions of whooping-cough in London are longer and more variable. Two years, more or less, intervene between the waves. The height of the wave in all these diseases is often seen to bear an inverse relation to the swell. This is easily understood, for the more constantly and

severely a contagious disease is present in a community the less material will be left for the recurring explosions. But it deserves notice that for a series of years severe explosions are the rule, followed by another series in which they are much less marked. Compare, for example, the period 1838-1866 (Chart II., diagram 1) with the succeeding period.

These explosions, whatever may be their cause, are not, as a rule, determined by a recurring increase in the virulence of the contagium. Indeed, the case-mortality in epidemic years is often lower than in the years of minimum prevalence. In the years 1872 and 1894, for example, measles were epidemic in Hamburg, but the case-mortality in the former year was 2·74, and in the latter 3·4 per cent. These waves are local explosions for the most part, not brought about by increase of virulence. But every now and again in the case of measles and small-pox, and perhaps also in that of all contagious diseases, virulence waves intrude. Thus a severe epidemy of measles occurred in Hamburg in 1892; the case-mortality reached 7·3 per cent. This was no local outbreak. Its impulse is to be traced in diagrams 1 and 2, Chart II., in London and New York, and it was felt at Paris, and doubtless in other widely distant places. The epidemy of small-pox in 1871 was such a virulence wave, which swept over the greater part of the world. Nothing is really known of the causation of these virulence waves, nor much of the frequency of their occurrence.

The ordinary biennial wave of measles that appears in large towns is evidently connected in some way with the diminution of susceptible subjects in the intervening years, and the interval necessary for accumulating a fresh mass of material for a new explosion. The fact that the failure of the epidemic wave in one year is sufficient to alter the biennial rhythm is conclusive on this point. Another proof is to be found in the occasional lengthening of the interval after an unusually high wave, as in the year 1839, and again in 1845 (diagram 1, Chart II.). But in what way does this thinning of the ranks of the susceptible necessitate a more or less definite interval to elapse before a new outburst can take place? In other words, why should these diseases proceed by way of successive explosions, and not occur continuously? Why should the virus, which is never absent from a large town, not spread in the intermediate years (*see* Chart IV. p. 150) among those who had escaped in the previous epidemy and among those that are being constantly added by the natural increase of the population, instead of waiting for an accumulation of subjects in order to clear them all off at one stroke? Ransome, who has devoted much attention to this point, thinks that "all the facts would be accounted for if we suppose that these diseases can only become epidemic when the proximity between susceptible persons becomes sufficiently close for the infection to pass freely from one to the other. When an epidemy has cleared away nearly all the susceptible, it is only when the meshes of the network of communication are again sufficiently close for it to include all the susceptible persons in one great haul that it can return." It cannot be doubted that the intervals between epidemics have something to do with the diminished numbers and density of the susceptible, but this "density" explanation is not without its difficulties. If a definite density of the susceptible were required before a disease can become epidemic, can we suppose that anything like the same densities will be attained in the same time in places where the outbreaks occur at the same intervals? If, as is generally believed, measles are only communicable by a proximity so close as that afforded by personal intercourse—say a few feet or yards—the density of the susceptible can never be such as to enable the infection to pass from one to another. The fineness of the meshes of the net can only be relevant to the epidemic spread of the disease if it secures the inclusion of the susceptible automatically, so to speak, that is, by the infection passing from one to another, from point to point, apart from that accidental intercourse between the sick and the healthy which does not depend on given density. Perhaps if we knew more of the manner in which the contagion really spreads in epidemic times, some or all of these difficulties would disappear. We think it by no means improbable that the interval may have a relation to a temporary immunity conferred on a population generally —on those who have not been attacked, as well as those who have—during the previous outbreak; just as in the case of cholera a severe epidemy confers an immunity for three years on a community.

IV. Seasonal Fluctuations.—"All diseases occur at all seasons of the year, but certain of them are more apt to occur and to be exacerbated at certain seasons." This aphorism of Hippocrates applies especially to epidemic disorders. The seasonal prevalence of diseases due to non-obligatory parasites is determined, as a rule, by climatic conditions affecting their saprophytic growth. Apparent exceptions occur, and the season of greatest prevalence in some localities is not that in which the virus seems to have the best chance of multiplying outside man, but that in which it has the greatest facilities for diffusion. But peculiar local conditions may convert a summer or autumn disease into a winter or spring one by actually favouring the growth of its organism at these seasons. We have an instance of this in the case of enteric fever, which is least prevalent in Munich in October, the month when it attains its maximum in England and in most other countries, and

it is interesting to note that two out of the three epidemics of cholera that have visited Munich (1836-37 and 1873-74) have also fallen on winter. It is thought that the breeding-places of the virus of these diseases at Munich are situated at a considerable depth, and that the optimum temperature for the growth of the virus is found at the seasons in which they are actually most prevalent. These apparent exceptions do not invalidate but confirm the rule. All the diseases of this class are not in the same degree or in precisely the same manner influenced by climatic conditions. The cholera vibrio does not grow at a temperature under 60° F., and its optimum ranges from 80° to 104° F. This explains why epidemics of cholera in temperate climates are so generally restricted to summer and autumn, severe winter outbreaks being among the rarest of events. The vibrio is rapidly destroyed by drying, hence extensive epidemics do not occur during the long dry season in the Punjab and Central Provinces of India. The vibrio cannot multiply in very dilute nutrient solutions, hence the extensive floodings occurring at certain seasons in Bengal, Assam, and other countries are followed by a fall in the cholera wave. Another explanation of this recession of cholera during the season of floods is suggested by Woodhead. He holds that when the cholera bacillus in the fæces passes directly into a soil so damp that it holds insufficient oxygen to satisfy its saprophytic requirements it remains an anaerobic organism readily and rapidly killed; but when the depth of the drying zone is greater, the soil contains more air, the organisms multiply, become more hardy, resist putrefactive organisms, acquire saprophytic habits, and become more dangerous.

The bacillus of enteric fever is able to multiply at lower temperatures than that of cholera. It maintains its vitality for weeks when repeatedly subjected to a temperature below the freezing point. It is also much more resistant to drying than the cholera vibrio; these facts explain why typhoid fever is not so distinctly a seasonal disease as cholera.

The season of our home cholera is even more strictly regulated by meteorological states—among which temperature occupies the first place—than its Asiatic congener. As Sydenham says, "Cholera begins in August, and within the limited barriers of one single month runs its course." But outbreaks of choleraic diarrhœa, closely dependent as the disease is on a high temperature, may occur in mid-winter, and not only so, but the very lowness of the temperature is in these cases the cause of its appearance. Such epidemics have frequently happened in Altona on the Elbe, when the highly impure water derived from that river, after it has received the sewage of Hamburg, has been distributed to the population on account of very severe frosts having deranged the filtering apparatus.

As an epidemic disease dysentery is to be classed among the summer-autumn group. Of 705 outbreaks recorded by Hirsch, 528 occurred in summer and autumn, and only 14 in winter; but in many parts of India the disease, attacking as it often does those debilitated by the malaria of autumn, is most prevalent in winter. No disease is more dependent on temperature than yellow fever. In determining the seasonal evolution of cholera, humidity as well as temperature counts for much, but the fluctuations of yellow fever appear to be determined by temperature alone. As Hirsch shows, it only exists throughout the whole year in regions where the mean winter temperature does not fall below 68°-72° F., and in these it attains to epidemic diffusion only in the hot season. In higher latitudes, with an isotherm of less than 68° F., yellow fever occurs as an epidemy only in years when the temperature comes up to that of tropical regions, and then principally in the hot season. In places with a still cooler climate the disease occurs almost without exception in the hot season only. A fall in the temperature to, or near to, freezing point puts an end to an epidemy. This coincidence in the fluctuations of yellow fever with those of the thermometer stamps the saprophytic character of its microbe.

The seasonal relations of plague demand further investigation. The fact that the disease generally raged in Europe in summer and autumn and died out in winter, and that, on the other hand, it shows little tendency to invade distinctly tropical regions, seems to indicate that the growth or diffusion of the bacillus is inhibited both by high and low temperatures. It is said that its epidemic progress in Mesopotamia is checked by an air temperature of 86° F. and stopped by one of 113° F. (Payne). There is little evidence, however, that the temperature met with in those parts of India where it has been lately raging have in any way modified its epidemic evolution. The mean temperature at the height of the first epidemy in Bombay was 74°-76°; in Poona, 81°-85°; in Surat, 81°-91° F. The slight difference between the temperature of the month in which plague was at its height and that in which it began rapidly to decline in a particular locality, and the considerable differences in the temperatures at which it rose and fell in different places, forbid us ascribing any marked influence to climate in controlling its course. The climate of Bombay is never so high or so low as to affect its epidemic evolution. Other climatic conditions than temperature probably come into play (see *Brit. Med. Journ.* 23rd Dec. 1899).

Some of the contagious-infectious class of diseases attain their maximum and minimum with great regularity at fixed seasons in a given country, but at different seasons in different

countries. It is possible that in one country their seasonal prevalence may be determined by circumstances favouring their contagion, in another by those favouring their infection. No doubt these two factors—contagion and infection—are not of equal importance in respect to their spread in all countries, nor in the same country at all times. Diphtheria in most countries is at its minimum in the third quarter and at its maximum in the fourth or first quarter. In London, Baltimore, Alexandria, and some other places the minimum falls on the second and the maximum on the third quarter. In England the more prevalent the disease is, the more pronounced is the autumn rise, a circumstance which seems to indicate that in epidemic years infection plays a greater part in its spread than in ordinary years.

Scarlet fever differs entirely as regards the season of its maximum and minimum prevalence in different countries. In London it begins to increase in May, and its maximum falls with great regularity on October; and it is noteworthy that its season has remained unchanged from the days of Sydenham. In Berlin, Hamburg, Copenhagen, and the Netherlands the seasonal incidence of scarlet fever is similar to that of London, *i.e.* it is least prevalent in the second, and most prevalent in the fourth quarter. In Paris and in the United States its maximum and minimum are just reversed. The only explanation we can offer of this strange phenomenon is that in some countries infection, in others contagion, plays the most important part in its spread.

Small-pox and measles differ from all other epidemic diseases in respect to their seasonal fluctuations, and these practically correspond all over the world, and have remained unchanged from the earliest times. They were winter or spring diseases in the time of Rhazes; they are so now. They break out now in winter, now in spring. In one place winter outbreaks are more frequent; at another place spring epidemics are more common; and, as we shall presently see, the relative frequency of winter and spring explosions varies in the same place in different series of years. Chart III. represents the monthly distribution of cases of measles in Edinburgh for ten years. It conveys the impression that the disease occurs continuously all through the year, with two maxima, in April and December respectively, and two minima, falling the one in December, the other in January. But if we look at Chart IV., representing five of the ten years, it will be seen that distinct epidemics occur either in spring or in the late autumn or winter. This seasonal incidence of the disease (and it is the same in small-pox) is observed all over the world, in North America as in Europe, and at corresponding seasons in the southern hemispheres.

The explanation of the epidemic prevalence of measles and small-pox in winter and spring is not to be sought for in the action of meteorological influences on their contagia, but in the effect of season on the susceptibility of the body generally, or on the vulnerability of the upper air-passages. The winter increase of small-pox all over the world coincides with the first notable fall in the temperature. It occurs in October in London and New York, in November and December in India, and at corresponding seasons in the southern hemisphere. In London small-pox and measles begin to increase in the fortieth, and bronchitis in the forty-first week. From this we infer that the same climatic conditions which determine respiratory affections also favour the infection of measles and small-pox. The change from winter to spring once more upsets the physiological balance, and the great vicissitudes of temperature peculiar to this season render the system more susceptible to the infection. This explanation is in harmony with what we know of the action of climate in determining the seasonal prevalence of diseases due to non-obligatory parasites. Climate in this case acts by preparing the soil for their growth. It is the same in the case of the obligatory parasites. The soil of the obligatory parasite is the human body, and climatic conditions which render the body more suited for its reception and growth at one season than another determine its seasonal prevalence.

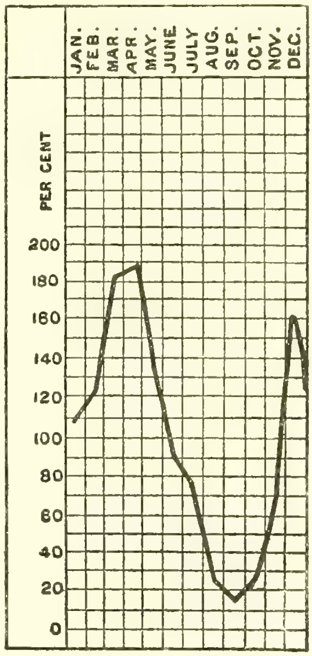


Chart III. Measles, 1880-89 (Edinburgh).

An important peculiarity in the seasonal fluctuations of small-pox and measles is that the mildness or severity of an epidemy has the effect of changing its seasonal incidence. It will be seen from Chart V. that mild epidemics of measles fall on the fourth quarter, and severe ones on the second quarter.

A somewhat similar change takes place in the seasonal prevalence of small-pox, as will be seen from Chart VI., the maximum in epidemic years being transferred from the first to the second quarter.

It would appear that virulent waves both of measles and small-pox occur most frequently in

spring,[?] but how this should be the case has still to be discovered.

The seasonal movements of whooping-cough are different from those of small-pox and measles. In Europe generally, this disease is most prevalent in the first or second quarter, in the United States in the third quarter.

Influenza is so erratic in its visitations that there is little time to study its seasonal evolution in any particular locality. It has been thought by some that its prevalence is unaffected by season. This is a mistake. A very full record of epidemics of influenza has been compiled by Hirsch. It will be found from a study of these that epidemics of influenza for the most part begin in winter, but as they spread they appear in a particular country earlier or later in the year, according to their distance from their point of origin. Of 14 Russian epidemics that spread to other parts of Europe, 11 began from November to January. Of 240 outbreaks in different regions in the northern hemisphere 84 occurred in the first, 45 in the second, 46 in the third, and 67 in the fourth quarter. We are justified, then, in classing influenza among diseases of the cold season, although it often spreads in the warmest weather. Dengue, which some have thought to be a form of influenza, differs essentially in its seasonal characters. It is not only a disease of warm countries but of the warm season. It is most prevalent in summer and early autumn.

The seasonal fluctuations of malaria, from what is known of its causation, may be inferred to be closely connected with the seasonal activity of the insects in which the parasite develops; but this requires further investigation. In the meantime it is to be noted that malarial fevers begin to increase in Northern and Central Europe in February, and attain their maximum in April and May, a season when insects of the mosquito species are little in evidence.

The laws regulating the seasonal fluctuations of epidemic diseases may be thus stated: Diseases of the infectious group are most prevalent in the seasons when the climatic conditions favour the saprophytic growth of their organisms. The seasonal fluctuations of the contagious group are determined by meteorological influences predisposing the body to infection. Both of these factors are in operation in regulating the seasonal movements of diseases belonging to the contagious-infectious group.

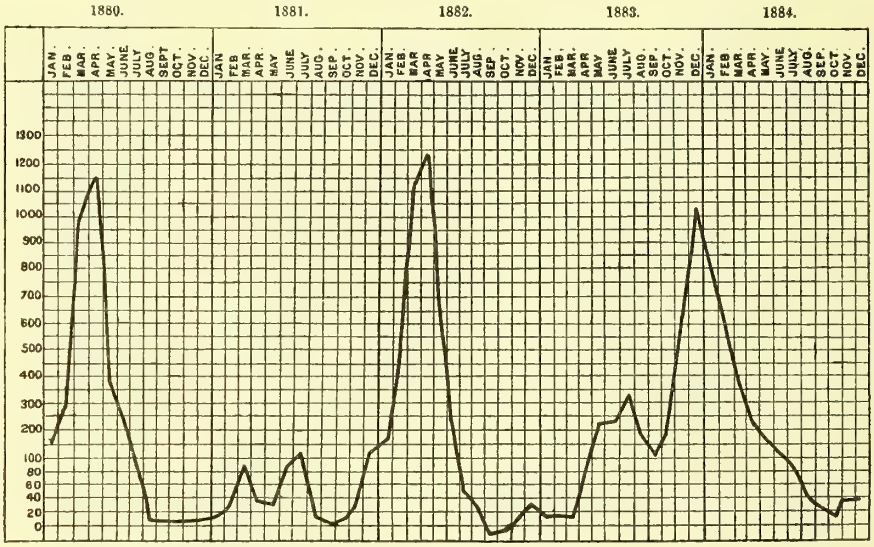


CHART IV.—Monthly intimations of Measles (Edinburgh) for the several years 1880-84. (Littlejohn.)

V. OSCILLATIONS.—The evolution of an epidemy is marked by minor and major oscillations. Small explosions at irregular intervals of days are doubtless caused by a spark of the infection falling accidentally upon an accumulation of combustible material. The major oscillations are more regular, and have been shown by Ransome to correspond to a large extent in places so far apart as London and Manchester. They appear to be caused by weather.

THE LAW OF ANTICIPATION.—Sydenham remarked that "if fevers, continued or intermittent, appear unnaturally early, the season that follows will be exceedingly favourable to the development of epidemics." We have satisfied ourselves that this law holds good as respects intermittent fevers in India. In years when malarial fevers are epidemic they invariably begin to increase before their usual period. But this law is of much wider application. An anticipation in the usual period of rise is observed in the case of most contagious diseases in this country in epidemic years. The rise in scarlet fever, for example, begins a month

earlier in years when it is epidemic than in non-epidemic years. This is a point of considerable practical importance, inasmuch as when a rise is observed in the cases or deaths from any of these diseases at the period when they are

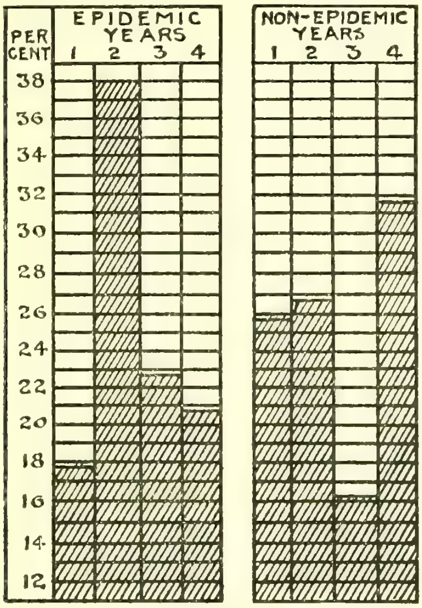


CHART V.—Quarterly incidence of Measles (London) in epidemic and non-epidemic years.

usually at their minimum, precautions should be taken against an impending outbreak.

Associations and Antagonisms of Epidemic Diseases.—The physician who attended John Evelyn for small-pox in Geneva in 1646 justified his having bled his patient before the appearance of the eruption by saying that but for the bleeding the distemper would have turned to plague or spotted fever. Few doubted at that time that diseases so widely different as plague, small-pox, and spotted fever could be converted one into the other, under changing constitutions of the atmosphere, or as a result of treatment.

It was another common belief in the Middle Ages, supposed to be supported by observation, that an unusual prevalence of small-pox, measles, spotted fevers, or agues heralded an outbreak of plague. Concoregio (1438) says: Multiplicantur autem præcipue variolæ, et sic ad experientiam visum est, in anno, qui præcedit pestilentiam futuram de proximo. Et est tanquam signum prognosticum ejus, quando ultra consuetum veniunt." In the same way Bacon affirms that "the lesser infections of small-pox, purple fever, and agues in the preceding summer, hovering all winter, do portend a great pestilence in the following summer." Spotted fevers of unwonted malignity preceded the outbreaks of plague in London in 1625 and 1665, the plague of Nimeguen in 1636, that of Naples in 1656, and of Moscow in 1771; and it must be remembered that spotted fevers and agues were then so common that one or other was almost sure to precede any outbreak of plague.

We have already alluded to Haeser's doctrine, founded, as he believes, on historical evidence, that the exanthemata, diphtheria, and dysentery prevail together for a long series of years, and then give place to the typhus class of maladies. The scattered records of disease before the registration era form, we think, an insecure basis for this deduction.

Since registration began in England we have had four groups of years marked by an extraordinary prevalence of small-pox, scarlet fever, and measles, and, curiously enough, these were on each occasion—contrary to Haeser's doctrine—associated with typhus. These periods were 1838-40, 1847-48, 1862-64, and 1869-72. The first three of these periods were times of much distress, which may explain the prevalence of typhus, but the considerable recrudescence of the disease, which took place in 1869-71, cannot be so accounted for. The great prevalence of small-pox, scarlet fever, and measles in these years seems to point to some atmospheric conditions which favoured their spread.

Sydenham held that as one nail drives out another, so one epidemic disease displaces another; but experience proves that two or more epidemic maladies, affecting the same or different age-periods, may prevail in the same place and at the same time. Witness the frequent association of epidemics of typhus and small-pox, of typhus and dysentery, of diphtheria and scarlet fever and measles. Nor does an epidemic disease extinguish for the time common forms of sickness, as some have asserted. Thucydides remarked that the year in which

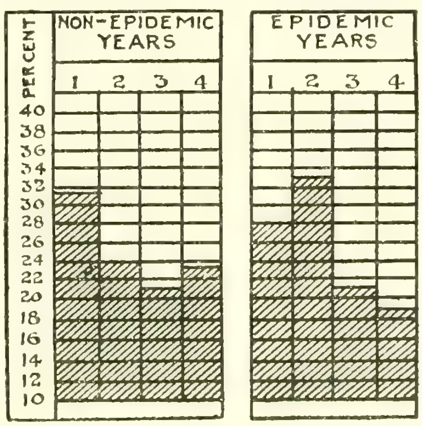


CHART VI.—Quarterly incidence of Small-pox (London) in non-epidemic and epidemic years.

the plague raged in Athens was notably free from other diseases; and Sydenham says that the year of the great plague of London was in other respects healthy, "so that all who kept clear of the plague never were better than then." When a large portion of a population

is carried off by a plague, fewer are left to die of other diseases. Plague may also prevail in an otherwise healthy year, but there is no proof that the existence of a plague suppresses other maladies.

One or two instances of apparent antagonism between epidemic diseases must be admitted, although they cannot be explained. When epidemic malaria overran New England from 1864 to 1884, it was noticed that as the malarial wave advanced in Connecticut typhoid fever receded. From causing four or five hundred deaths in a year, it so decreased in prevalence that in one of the malarious years the deaths from typhoid fever fell to one hundred and fifty-nine. As malaria disappeared typhoid fever came once more to the front. It has also been observed in some instances that epidemies of malarial fever have suddenly ceased when cholera appeared. But the reverse has also been observed. It is recorded that intermittent fever disappeared entirely from Marienwerder (where it was previously epidemic) on the cessation of cholera in 1831, and only reappeared with the return of cholera in 1849. It did not, however, disappear along with the cholera as before, but remained the predominating sickness in that locality until 1856, when it again diminished in frequency (Hirsch). We have no satisfactory explanation to offer of these strange associations and antagonisms.

Evolution and Involution of an Epidemy.—On examining the course of the great epidemy of small-pox in England and Wales in 1837-39, Farr found that the deaths in the first two quarters of its progress increased at a uniform rate of 30 per cent. From the third to the fourth quarter, the increase was only 6 per cent, after which the numbers for a short time remained stationary, like a projectile at the summit of its curve. The deaths then decreased through six quarterly periods at successive ratios of 5, 10, 15, 20, 26, and 31 per cent. Vague notions have been, and are, entertained that some such law will be found to apply to all contagious diseases. It has been conceived that an epidemy, like a projectile, describes a definite trajectory, so that, given the ratios of increase at two points in its course, its further progress could be calculated.

The evolution of an epidemy follows no fixed law. Indeed, the ratios of increase and decrease of the *same* disease differ in different outbreaks in the *same* locality. That this must necessarily be the case will be evident if we reflect that the course of an epidemy—say of small-pox or measles—depends on a number of factors that are never present and operative in the same degree in any two outbreaks. These factors are: (*a*) the spreading-power of the virus, which is a variable quantity; (*b*) the number of centres from which the epidemy starts; (*c*) the facilities for the spread of the contagion, dependent on the number of the susceptible, their aggregation, and the degree of intercourse among them; (*d*) the seasonal influences, which accelerate or retard its spread. The last-named factor has an important effect on the ratio of increase. Scarlet fever, for example, is normally at its minimum in the second quarter. During an epidemy, the deaths in the second quarter, it is true, exceed those in the first, but the rate of increase during that quarter is invariably slowed by inhibiting seasonal influences.

These considerations would lead us to anticipate considerable diversities in the evolution of the same disease. But every epidemy of a contagious disease in a large community has, nevertheless, its period of rise, its fastigium, and its period of decline. Normally the ratio of increase is an accelerating one up to a certain point, when it becomes retarded, and the rate of increase in the number of attacks then becomes less and less until the fastigium is reached. The decline now commences. At the beginning it is slow, then it proceeds more or less rapidly for a time, and slows down again, as the disease approaches its sporadic level.

How are we to account for these stages? We can readily understand that, once an epidemy has been set agoing, it will advance more and more rapidly as the contagium becomes more and more multiplied and diffused. As the numbers of the susceptible decrease and their density diminishes, an arrest of the rate of spread will take place, and at a certain point the tide must turn, and the number of attacks become fewer and fewer until the outbreak subsides. Does this mechanical theory of numbers and density account for the trajectory described by an epidemy? The retardation in the rate of spread is not to be accounted for solely by the reduction in the actual number of the susceptible, for the number attacked subsequent to the slowing of the ratio of increase is greater than that up to the point when the retardation begins. But the diminished density of the susceptible has to be taken into account; for although the thinning process is in operation from the beginning of an epidemy, and is to some extent counteracted by the increasing diffusion of the contagium, yet when it has reached a certain point the diminished density must have an effect in slowing the rate at which attacks proceed. Other factors modify the course of an epidemy, but the governing factors are the numbers and density of the susceptible, and, as has already been said, many who have not passed through the disease may acquire a temporary insusceptibility. But the question here arises, Does the infective agent undergo changes of virulence during the progress of an epidemy?

The case-mortality varies according to the different phases of an epidemy, as will be seen from the following figures, which give the number of cases of measles, the deaths, and the

percentage of deaths to cases in thirteen four-weekly periods, based on the Hamburg returns for the sixteen years 1879-94. The deaths in one period are credited to the cases in the preceding period:—

Four-Weekly Periods

	1.	2.	3.	4.	5.	6.	7.
Cases	4017	3568	3525	3564	6888	10,721	9284
Deaths	166	136	180	255	444	508	256
Percentages	4·1	3·9	5·1	7·2	6·4	4·7	2·8

	8.	9.	10.	11.	12.	13.
Cases	4695	2382	2637	4094	5638	5359
Deaths	166	101	156	205	213	167
Percentages	3·5	4·2	5·9	5·0	3·8	3·1

The case-mortality will be seen to rise in the third period, when the cases are at their minimum. A further rise occurs in the fourth period, when the increase in prevalence has begun, but is still insignificant. With the first epidemic bound, in the fifth period, the case-mortality begins to fall, and continues falling rapidly as the epidemy attains and passes its fastigium in the sixth and seventh periods. A reverse movement then sets in, and the case-mortality rises until the minimum prevalence is reached in the ninth period. As the winter epidemic extension begins in the tenth period, the case-mortality shows a further rise, but no sooner does the disease become widely prevalent than the case-mortality again begins to fall. In short, the virulence of the disease, measured by the case-mortality, appears to bear throughout pretty much an inverse relation to its diffusion.

Now the question arises, is the increase and decrease of the virulence of the contagium a cause or a consequence of the increase and decrease of prevalence in the varying phases of an epidemy? It must be remembered that virulence and spreading-power are not necessarily related, for the case-mortality is often low in rapidly and widely-spreading epidemies. Besides, a diminished case-mortality may mean either an increased killing power of the virus, or an increased resistance on the part of those attacked, so that fewer succumb to the disease. Upon the whole, it appears probable that the case-mortality becomes lower as a consequence of the wider diffusion of the disease. The question how this occurs cannot be fully discussed here.

There is a tendency at the present day to refer the evolution and involution of epidemies exclusively to changes in the virulence of the contagium. It is assumed that at the outset of an epidemy only the more susceptible are attacked, and that in passing through these the virus gains in intensity and spreading-power. The turn of the less susceptible then comes, in passing through whom the virus becomes attenuated. The virulence of a germ is undoubtedly exalted or attenuated according as it is cultivated in susceptible or resistant bodies, but it is not so obvious why the virus at the early stage of an epidemy, when it is by no means deficient in potency, should select only the more susceptible for attack. It is more probable that the wide diffusion of the virus in a community increases the resistance of the susceptible, and thus brings about an attenuation of the microbe.

The decrease in the virulence of the contagium (granting that the decreased case-mortality indicates a decreased virulence) may be brought about in other ways than by merely passing through more resistant subjects. When the contagium, for example, is widely diffused, it is reasonable to suppose that the infection is, in many cases, propagated by fomites. The virus, subjected for a varying time to the influence of light and air, will become attenuated, and thus give rise to a milder type of the disease.

The course of an epidemy of one of the infectious diseases is much more uncertain and irregular than that of a contagious disease. Its outbreak, extent, fluctuations, and duration are largely dependent on circumstances that are variable and contingent, such as the sanitary state of a town into which the infection is introduced, the water-supply, and such like.

An accidental contamination of milk or water by the virus of cholera, scarlet fever, or enteric fever often gives rise to a sudden outburst, which rapidly subsides if the contamination is not renewed. No distinct periods of rise, fastigium, and decline are to be looked for in explosions of this kind. They present the characters of a wholesale poisoning rather than those of an epidemy. Further, when an infectious disease assumes an epidemic character, its course may be modified or arrested at any stage by a change in the weather unfavourable to the saprophytic growth of the virus, as in the case of yellow fever, or from the virus being deprived of its vehicle of diffusion, as when, during an outbreak of cholera, an infected water-supply is closed.

Another circumstance which contributes to make the evolution of epidemies of infectious diseases irregular is the great variability in the virulence of their contagia. A cholera outbreak may run the whole or a part of its course as a severe diarrhœa. Enteric fever may present itself under the guise of a febrile gastro-intestinal catarrh. Plague in the course of a single epidemy may assume different forms with varying diffusive powers. All these circumstances cause the greatest differences in the course of an epidemy of an infectious disease at a particular time and in a given locality. But if we take the course of an outbreak in a whole country for a limited period, or in a given locality, over a number of years, these irregularities disappear, and the epidemic evolution of an infectious disease will be found, on the large scale and in the long run, to be governed by the conditions which promote or hinder the saprophytic growth of the contagium.

Epidermidolysis.—Loosening of the attachment of the epidermis, leading to its exfoliation.

Epidermidoses. — Diseases affecting the epidermis, including the keratoses (*e.g.* ichthyosis), the chromatoses, and the acanthoses.

Epidermis.—The cuticle or scarf-skin. *See* SKIN, ANATOMY AND PHYSIOLOGY (*Epidermis*).

Epidermolysis.—That morbid state of the skin in which bullæ form as a result of slight causes (*e.g.* irritation); the tendency may be hereditary (*Epidermolysis bullosa hereditaria*). *See* PEMPHIGUS (*Epidermolysis*).

Epididymis. *See* SCROTUM AND TESTICLE, DISEASES OF (*Anatomy, Abnormalities, Encysted Hydrocele*, etc.); TUBERCULOSIS (*Morbid Anatomy, Epididymis and Testis*).

Epididymitis. — Inflammation of the epididymis. *See* SCROTUM AND TESTICLE, DISEASES OF (*Epididymitis*); HYSTERIA (*Disorders of Genital Organs*); UNDULANT FEVER (*Symptoms, Sexual System*); URETHRA, DISEASES OF (*Gonorrhœa, Complications*).

Epigastralgia.—Pain in the epigastric region.

Epigastric Pulsation. *See* BRONCHI, BRONCHITIS (*Physical Signs*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Physical Examination, Inspection, Palpation*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Aortic Incompetence, Inspection; Mitral Stenosis, Inspection*); PULSE (*Heart Movements, Epigastric Pulsation*).

Epigastriocele. — A hernia in the epigastric region.

Epigastrium. *See* ABDOMINAL ANEURYSM (*Symptoms, Epigastric Tumour*); ABDOMINAL TUMOURS, DIAGNOSIS (*Tumours of Abdominal Contents*); LIVER (*Abscess, Sub-hepatic*).

Epigastroschisis.—Defective formation of the abdominal walls in the epigastric region.

Epigenesis.—The embryological theory, first indicated by Harvey and later established by Caspar F. Wolff, according to which the complex embryo arises from a relatively homogeneous germ by gradual differentiation of parts; it was opposed to the idea of the pre-existence of germs.

Epiglottis. *See* LARYNX, EXAMINATION OF (*Laryngoscopy, Appearances of Epiglottis*); LARYNX, CHRONIC INFECTIVE DISEASES (*Laryngeal Phthisis*); LARYNX, CHRONIC INFECTIVE DISEASES (*Lupus*); PHYSIOLOGY, RESPIRATION (*Voice*).

Epignathus. *See* TERATOLOGY. — A teratological type in which a rudimentary fœtus is attached to the jaw (usually the upper) of an otherwise normal child; the parasite usually protrudes through the mouth of the autosite.

Epilation.—The removal of hairs by plucking or pulling them out; depilation.

Epilepsy.

See also ALCOHOLISM (*Epileptic Fits*); ATHETOSIS (*Following Epilepsy*); BRAIN, CEREBELLUM, AFFECTIONS (*Atrophy, Diagnosis*); BRAIN, TUMOURS OF (*Diagnosis*); BRAIN, CYSTS (*Porencephaly*); BRAIN INFLAMMATIONS; BRAIN, SURGERY OF (*Depressed Fragments of Skull*); BRAIN, SURGERY OF (*Epilepsy*); BROMINE; CATALEPSY (*Etiology*); CHOREA (*Complications*); COLOUR VISION (*Disturbance of Colour Perception, Causes*); CONVULSIONS, INFANTILE; DECHLORINATION; GENERAL PARALYSIS (*Diagnosis*); HEREDITY (*Epilepsy in Guinea-Pigs*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Bradycardia in Epilepsy*); HYPNOTISM (*Uses*); HYSTERIA (*Epileptic Period, Diagnosis*); HICCOUGH; LARYNX, BENIGN GROWTHS (*Removal has cured Epilepsy*); MALINGERING; MEMORY IN HEALTH AND DISEASE (*Defects*); MENTAL DEFICIENCY (*Varieties, Developmental, Epileptic*); MIND, EDUCATION (*Epileptic Children*); MYIASIS (*Intestinal, Symptoms*); NAILS, AFFECTIONS OF (*in Diseases of Nervous System*); NOSE, NASAL NEUROSES; PUBERTY (*Disturbances of Health*); SENILE INSANITY (*Neuroses, Epileptoid*); SLEEP, NORMAL AND MORBID (*Morbid Dreams in Epilepsy*); SKIN, PIGMENTARY AFFECTIONS; TABES DORSALIS (*Epileptiform Seizures*); TETANY (*Diagnosis*); TRADES, DANGEROUS (*Lead-Poisoning, Diagnosis*); TYPHOID FEVER (*Complications and Sequetæ*); UNCONSCIOUSNESS (*Epilepsy*); URINE, PATHOLOGICAL CHANGES (*Albuminuria after Epilepsy*).

DEFINITION.—Epilepsy is a disease characterised by the recurrence of paroxysmal seizures of greater or less severity, the dominant feature of which is a sudden loss or impairment of consciousness, and which is not necessarily due to any obvious organic disease of the brain, its membranes, or its blood-vessels.

The feature whose presence is necessary to establish the diagnosis of epilepsy may consist of anything, from a momentary blurring up to complete abolition of consciousness. The convulsive element is not essential; if it occurs it may vary from a slight involuntary deviation

of the eyes up to spasm of the whole muscular system.

But there are several forms of coarse brain disease which may give rise to convulsions not differing from those of idiopathic epilepsy. Such are thrombosis of the cerebral blood-vessels, both in the old and in the young; chronic meningo-encephalitis of specific nature, old-standing foci of hæmorrhage, and in adults, intracranial new-growths, during their incipient stage.

Etiology.—(*a*) In its relation to the total population difficulties arise in reaching satisfactory statistical results as to the prevalency of epilepsy. First, no census for this purpose has been taken in the United Kingdom, while those made abroad have been only partial; and, secondly, epilepsy is so closely related in the early years of life to idiocy and imbecility, and in the later years to insanity, that it is almost impossible satisfactorily to separate them for statistical purposes. A few facts may be stated as to the estimated number of adult epileptics. The estimate is given as 1 per 1000 in Germany, 2 per 1000 in the State of New York, and 2·5 per 1000 of total population in the Canton Aargau in Switzerland. Of 10,000 young men who presented themselves for military duties in Italy, 11·5 were rejected owing to epilepsy, the proportion being just over 1 per 1000. The probable proportion, therefore, of adult epileptics to the total population averages from 1 to 1·5 per 1000 in the European countries. In the Australasian colonies it has been found that the proportion was lower than that estimated for Europe and America. For example, in Victoria there appears to be 1 epileptic in every 2715 of population—approximately, an average of ·25 per 1000. These estimates embrace all cases of epilepsy, including asylum cases. It may, therefore, be considered a fair average when it is stated that in Great Britain there is roughly 1 epileptic in every 1000 of the population.

(*b*) That epilepsy should have existed in the parents or progenitors of an epileptic person does not seem to be essential. A tendency to one of the neuroses, more especially insanity and alcoholism, is sufficient. The statistics of those who have studied similar heredity approximate with a sufficient amount of uniformity to be of value. Thus Reynolds found it in 31 per cent of his cases, Gowers in 35 per cent, Berger in 32 per cent, and Echeverria in 27 per cent. It has been pointed out that when an inherited tendency exists, the female members of the family suffer more frequently than the males. Bouchet has shown the relation of parental epilepsy upon the incidence of convulsions in the offspring by referring to 58 children born of 14 epileptic mothers. Of these, 37 died in infancy from convulsions, and of the remaining 21, 7 became epileptic.

The oft-mentioned relation between phthisis and epilepsy appears to be of the nature of a coincidence, as it is not greater than amongst those who are not epileptics.

(*c*) The age of the individual in relation to the incidence of epilepsy is clearly determined by statistics. The commonest period for the commencement of epileptic fits is during the second decade of life. Of 1450 cases collected by Gowers, 39 per cent commenced during this period; of 995 cases collected by Hasse, 36 per cent; and of 980 cases by McLane Hamilton, 27 per cent. In the remainder of Gowers' cases there were 28 per cent in the first decade, 14 per cent in the third, and 5 per cent in the fourth decades. During the second decade the years from thirteen to seventeen inclusive are most favourable to the onset of epilepsy. Reynolds pointed out that when there was a marked hereditary influence, the disease began somewhat earlier than in those in which no such history was obtained.

Many epileptics give a clear history of convulsions in infancy. In some the infantile convulsions have never entirely disappeared, and in them a direct connection may be traced between the early convulsive seizures and the subsequent epileptic attacks. In others, however, a definite period intervenes between the subsidence of the convulsions and the onset of true epilepsy about the time of puberty. Hence the history of so-called "teething fits" is not at all uncommon amongst those who, towards the end of puberty, become epileptic.

In about one-third only of the total number of cases of epilepsy is the presence of an exciting cause obtained. With a view to associate the origin of their malady with some definite causation, many conjectural circumstances are narrated by the patient or his friends, most of which have little or no direct influence. Of the causes usually assigned, the commonest is *mental emotion*, such as worry, fright, and prolonged anxiety.

Traumatism, for instance, blows, falls, or kicks upon the head, much more commonly leads to convulsions of the Jacksonian type, from local injury to the brain, than to the production of true epilepsy. But whether cerebral injury is due directly to traumatic causes, or arises from so-called idiopathic conditions, such as infantile hemiplegia, convulsions may eventually ensue indistinguishable from those of true epilepsy (*see* "Convulsions," "Brain, Surgery of"). These are, however, more local in their commencement, and affect one side of the body more than the other.

Of the exanthemata scarlatina is the most common cause, apart from nephritis or cerebral thrombosis arising from middle ear disease. Typhoid fever and measles are less frequent agents.

There is no clear evidence that secondary syphilis is a factor in the causation of true

epilepsy. The acquired form of this disease occasions convulsions of a Jacksonian type, from characteristic vascular and gummatous lesions; though the resulting brain injury may in some cases give rise to fits indistinguishable from true epilepsy. Epilepsy arising in the subjects of inherited syphilis is more probably due to a lowered nervous vitality than to a direct effect of the syphilitic poison.

Masturbation is a symptom of the disordered cerebral state associated with some graver forms of epilepsy, and should not be regarded as a causal agent.

Alcohol is one of the commonest factors in the development of epilepsy in later life. Fürstner found that 31 per cent of his cases of delirium tremens were epileptic ; and Moeli obtained a larger percentage, viz. 36·4 per cent, though in the alcoholic insane only 10 per cent were epileptic. The alcoholic tendency and epilepsy are probably both the result of an inherited neurosis, and do not stand to each other in the relation of cause and effect.

Symptoms.—The epileptic seizure may be studied under three headings :—

I. *Le grand mal.*—This is the common or typical form of epilepsy, from which the disease originally took its name—the "falling sickness." It is characterised by a sudden and complete loss of consciousness, in which the patient falls down, and universal convulsions.

2. *Le petit mal*, consisting of a momentary loss or obscuration of consciousness without evident muscular spasm. It is usually in connection with this form that the extraordinary acts known as epileptic automatism are performed.

3. *Le petit mal* with slight convulsion, the "epilepsia mitior with evident spasm" of Reynolds, an important group standing midway between the big and the little seizure, important chiefly owing to the localised character of the spasm, in which respect it resembles the convulsions of organic cerebral disease.

(1) *Le grand mal.* — In considering the symptoms which precede an epileptic seizure, it is necessary to distinguish those which occur some days or hours before the attack—the prodromal symptoms—from those which immediately usher it in, the warning or "aura."

There is great variation amongst the *premonitory symptoms.* In those cases which present them they are usually of a mental character. Those who live with epileptics are able to tell precisely when the individual is likely to have a fit. On the one hand, some epileptics show abnormal moroseness, and a morbid irritability leading to frequent quarrels with their fellows, or a tendency to wander about in a depressed manner. Again, others show a liveliness which is quite unnatural to them; while in others distinct delusions may give indication of the approach of the fit. Headache, giddiness, and loss of appetite may take the place of the mental phenomena just described.

The "warnings" or "auræ" of epileptics are numerous, and their clinical value is often great, in that they point to the particular area of the brain in which the discharge commences. They may be described according as they belong to the sphere of motion, common and special sensations. Amongst motor auræ are many curious, co-ordinated actions, such as running forwards, or backwards, or turning round; one patient ran rapidly round the table and then fell down in a fit; another would stoop down and peep under the sofa (Reynolds); another would rapidly retrace his steps and then fall down (Gowers). Local motor phenomena, such as a definite muscular spasm in face, leg, or arm, though usually characteristic of organic cortical disease, is found in the variety of epilepsy designated "epilepsia mitior" with slight convulsion.

In the region of common sensibility numerous abnormal sensations are described : tingling or numbness in the extremities; headache; a sensation as of something rising from the stomach (epigastric aura); giddiness, and feelings referable to the head are also frequent.

Auræ involving the special senses are of great importance, as they indicate the commencement of the discharge in a special sense cortical area. There are a few rare cases on record in which a cerebral tumour occasioned a special sense aura associated with a "dreamy state."

The *fit* is, for purposes of description, commonly divided into three stages, which, however, fuse imperceptibly into one another.

First, there is the stage of *tonic contraction* of the whole muscular system associated with loss of consciousness and the "fall" of the patient. The tonic convulsion is usually more marked on one side, so that the head and eyes are turned to one side; the body may be actually rotated upon its long axis, and the limbs of one side are frequently more convulsed than the other; there is fixation of the chest from spasm of the intercostal muscles and diaphragm, this being accompanied by a groaning sound of characteristic nature, the "epileptic cry"; pallor of the face, followed by a congested appearance, with dilatation of the pupil and diminution in the force of the pulse, the whole period lasting about half a minute. This stage quickly passes into the second or stage of *clonic convulsion.* Here the muscles are jerked violently; the tongue may be protruded between the teeth and bitten, the bladder and rectum evacuated, and the eyeballs rotated in all directions; respiration is impeded by the irregular action of the respiratory muscles; the patient foams at the mouth, and the face becomes livid, and the veins of the neck engorged. In a severe fit death seems imminent from asphyxia, but when events appear

to be at their worst a slackening in severity occurs, and the *stage of resolution* is heralded by a return to consciousness. The involuntary convulsive movements are replaced by those of a voluntary nature, so that the patient may alter his position by turning round or by sitting up; on the other hand, movements of a hysterical character may occur; or the patient may struggle and fight so that several attendants are required to hold him down. All these, however, pass off, and the patient falls into a deep sleep.

The first stage may be said to last from five or ten seconds to half a minute; the second from one to two minutes; the third from five to ten minutes, according to the nature of the movements. The post-epileptic sleep may last for an hour or two, but sometimes the patient is sleepy and dazed for the remainder of the day.

There are a few points to which closer attention may be given.

The pupil, which sometimes shows a preliminary contraction, is usually dilated during the stage of asphyxia.

The pulse during the second stage becomes tense and increased in frequency.

The epileptic seizure is usually followed by severe headache, of which complaint is made on awakening from the period of sleep.

Immediately after the fit the reflexes may be abolished, but this temporary state is followed by an increase of the knee-jerks, and an ankle clonus may be obtained.

The temperature may be raised one or two degrees during the epileptic convulsion. In the event of a status epilepticus supervening the temperature may rise to hyperpyrexia.

Vomiting may follow an epileptic fit; in some cases it is a constant feature. When it occurs it forms an element of danger owing to the risk of choking.

The secretion of urine is copious, but in other respects it does not differ from normal.

(2) *Le petit mal;* a momentary loss of consciousness without obvious muscular spasm, a condition to which patients usually apply the terms "fainting attacks," or attacks in which they feel "dazed," or of "sensations," or of temporary loss of memory; or the friends may give an account of attacks in which the patient "looks queer." These seizures may last from one to two or five seconds. Should the person be conversing, he suddenly stops speaking, his face becomes pale, the pupils dilate, and the expression assumes a "blank" appearance. These are only momentary, and conversation is resumed as if nothing had happened. If on the other hand the patient is walking, he may be noticed to stagger slightly, or lean to one side—but he rarely falls, or lets an object drop from his hand.

These attacks give no warning of their approach, and pass off without any consequent dulling of the mind. On the other hand, the loss of consciousness may be accompanied by acts of an involuntary automatic nature. Hence it is in association more especially with attacks of *le petit mal* that the extraordinary actions known as epileptic automatism are observed. For instance, one patient sometimes turned out the contents of his pockets, while on other occasions he has been observed to pile the crockery on the dinner table, while acts of a distinctly aggressive nature are from time to time recorded. Another invariably took out his watch without knowing what he did, a very common automatic act, both after slight as well as severe fits, being the habit of undressing.

Slight attacks unassociated with automatic actions may exist for a long time and be overlooked. A not uncommon history is that of a person who has been suddenly seized with a severe epileptic fit; inquiry into the previous history shows that he has been subject also to momentary lapses of consciousness. Or another person, who occasionally suffers from attacks of *le grand mal,* may during the intervals of these seizures have frequent attacks of *le petit mal.*

(3) *Le petit mal* with slight convulsion, the "epilepsia mitior with evident spasm" of Reynolds.

This form of epilepsy is not uncommon, and, owing to the local nature of the spasm, the diagnosis from local cerebral disease may at the outset not be apparent. The convulsion, however, is always associated with temporary loss of consciousness. The spasmodic element may consist of a momentary conjugate deviation of the eyes, or of the head and eyes to one side; or the limbs may be chiefly involved, an arm may be thrown into slight tonic convulsion; or the convulsion may take the form of a hemiplegic jerking of the arm and leg. In the severer cases there is both a tonic and a clonic convulsion. In some cases a distinct "sensation," as if the limb were being moved, may precede or take the place of the actual convulsion. Another resemblance to Jacksonian epilepsy lies in the fact that the patient is often aware of the commencement of the attack, though unable to arrest it. These attacks are of short duration, several often occurring in close sequence, so that the total number recorded in one day may be large. This form of epilepsy was commoner in children and young people. The importance of its recognition lies in the fact that when it occurs during sleep it is a frequent cause of intermittent "wetting the bed." Owing to the temporary duration and slight nature of the convulsion the tongue is rarely bitten.

Having now briefly described the form of attack commonly associated with the term epilepsy, it is necessary to look shortly at several factors of importance in connection with the disease as a whole.

First, there is the onset and frequency of epileptic attacks. The time of onset having been referred to under the etiological heading of age, the manner of onset may be now mentioned. In some cases attacks of *le petit mal*, often unrecognised as epileptic, may precede for years the onset of a severe fit; in others, one attack may be seen without any recurrence for periods varying from a few months to as many years; or the onset may be in the form of a series of severe seizures to be followed after an interval by single fits. The common method of onset is for the initial seizure to be followed at periods of a week or two, or a month or two, by similar attacks. As already pointed out, intermittent "wetting of the bed" is highly suggestive of fits occurring during sleep.

Fits vary much in frequency, from one seizure once or twice a year or less, up to several dozen in one day. They occur both during waking and during sleep. A common time for their appearance is the period just before or just after rising in the morning. Some epileptics only have fits during sleep, which if slight may go unrecognised for a long time.

Epileptic fits occur both singly and in batches. Usually during the interval the patient is free both from sensations and subjective symptoms, and in many instances is perfectly capable of carrying on his work or profession. At times, however, the fits follow each other with such rapidity that the condition known as the *status epilepticus* occurs. Here the patient passes rapidly out of one attack into another; and this recurrence may be so prolonged as to reduce him to a condition of danger, and death not infrequently ensues.

The study of the periodicity of epileptic fits is of scarcely more than academic interest. At the epileptic colony it has been found that they are most frequent in the first hour after getting up in the morning; while fits were rare on going to bed. There were considerably more fits during waking than during sleeping hours, and more also in the house than outside.

Secondly, epilepsy has an important relation with the sexual history in women. It is well known that they are often more frequent about the time of the menstrual periods; and the onset of the disease in girls is usually associated with defect or delay in the starting of the catamenia. Its relation to pregnancy is variable. This condition has been found to increase their frequency on the one hand, on the other to diminish or entirely arrest their occurrence during the period of carrying. Fits have been known to occur for the first time at quickening. Epilepsy occurring during lactation, or the frequency of the fits being augmented during that period, are well-known facts. In one case fits were only seen during lactation following successive pregnancies, the weaning of the child invariably curing the disease.

Thirdly, the mental condition of epileptics requires special reference. It is an important feature of the disease. In this connection there have to be considered the effects produced upon the mind by the recurrence of epileptic seizures over a number of years, as well as the attacks of acute insanity which replace or follow fits in some persons. This is to be studied along with epileptic insanity described in another article. (p. 164).

(*a*) Either directly as the result of fits arising during infancy, or as the result of an inherited neurotic condition, epilepsy is closely associated with idiocy and imbecility. The cases are among the most difficult with which the physician has to deal. They are invariably dirty in habits, usually destructive, and always irritable. They can only be treated satisfactorily in the idiot asylums.

(*b*) The general mental features of epileptics. There seems to be no doubt that the constant recurrence of epileptic fits over many years leads, in the majority of cases, to mental impairment and deterioration. In some cases it is so slight as to be scarcely perceptible, the only indications being a blunting of the memory and a dulling of the finer feelings and emotions. In others a more pronounced state of dementia exists, characterised by loss of memory, a slowness of perception and of appreciation of ordinary circumstances; a want of initiatory power and inability to effectually carry out work, unless under direction; in others, hypochondriasis merging at times into actual delusional insanity of a temporary character; in others, irritability and impulsiveness to an abnormal extent; while nearly all epileptics show a morbid religious enthusiasm.

Attention has already been called to the fact that the onset of epilepsy, or of individual fits or batches of fits, is observed to be preceded for some days or longer by various mental aberrations, such as unwonted dulness, hypochondriasis, and loss of memory; an unusual sense of well-being and even at times distinct delusions, all of which may disappear under the influence of treatment.

It has been already stated that an epileptic fit is not uncommonly followed by symptoms of an hysterical nature. This is the common condition, and is not necessarily confined to the female sex. In other cases acute mental symptoms may follow an epileptic seizure, of which the most difficult to deal with, except in asylums, is *acute epileptic mania*,—a condition in which the person may become homicidal or suicidal, or subject to paroxysms of extraordinary fury and violence requiring restraint.

"Masked" epilepsy is the name given to outbursts of this nature when there is reason to suppose they replace epileptic seizures.

At the opposite extreme is the *acute epileptic dementia*, sometimes seen after a batch of severe

fits. Here the patient lies in semi-conscious state, requiring to be fed and nursed. In a few days he begins to show response to external conditions, becoming in some cases subject to temporary delusional insanity, but usually returning to the original mental condition.

An intermediate form of mental aberration, more easily dealt with and either replacing or following epileptic attacks, is found in temporary delusional insanity, sometimes accompanied by morbid eroticism, or by impulsiveness, or a condition in which "the patient is difficult to manage."

Pathology.—Epilepsy may be fairly stated to be a disease without a morbid anatomy. Various statements are found in the text-books as to the pathological conditions described in cases which have died from this affection. But no constant pathological lesion has been detected. Some of those alleged to be the causal agent, such as thickening of the bones of the skull, sclerosis of the cornu ammonis, and depressed spiculæ of bone, are coincidental circumstances. Others, such as engorgement of the cerebral sinuses, and minute hæmorrhagic extravasations and ecchymoses, are found in many other affections, and arise directly from the method of death, chiefly in coma.

In epileptic insanity, as described by Bevan Lewis, a fatty alteration in the nuclei of the cells of the second cortical layer, accompanied by cell vacuolation and disintegration, has been found; but it is scarcely to be supposed that such a condition forms the foundation of ordinary cases of epilepsy, as any prolonged vascular occlusion will bring about degeneration of the neuron.

The facts of experimental physiology clearly point to the cortex of the cerebrum as the region from which discharge of nervous energy takes place; but what is the nature and cause of the periodical hyper-excitability which leads to an epileptic fit is entirely of a speculative character. That the clinical phenomena which usher in one attack indicate the locality from which the discharge originates is now a well-established doctrine. From this spot the discharge spreads, so that in a severe epileptic seizure the whole musculature of the body is eventually thrown into convulsions. Hence it may be concluded that in cases without convulsion, or in those in which sudden loss of consciousness is the first symptom, that part of the cerebral cortex which is related to the mental processes is primarily involved. If beginning with a definite movement of a limb, the origin is in the cortical motor zone in which the extremity is represented. In cases with a special sense-aura it is generally held that the discharge begins in the cortical area under consideration, as direct evidence has been obtained from the few cases submitted to pathological examination, in which a tumour implicating the areas subserving taste and smell gave rise to convulsions preceded by "auræ" of those special senses.

Diagnosis.—The diagnosis of epilepsy is in many cases simple, in others the difficulty is great, more especially if the attack is being described rather than observed. An affirmative answer to one of the three following questions would usually characterise the "fit" as epileptic. During the attack—

(1) Did the patient fall down suddenly unconscious?
(2) Was the tongue bitten?
(3) Was urine passed involuntarily?

But a negative reply to these questions does not deny the possibility of epilepsy. None of these features are found in the variety known as *le petit mal*, or in the so-called epileptic automatism. For the diagnosis of these reliance should be placed upon a consideration of all the facts of the case, and chiefly upon automatic acts, these being absolutely diagnostic of epilepsy; but it is often difficult to differentiate between *le petit mal* and a "faint" of syncopal nature. The chief diagnostic point lies in the suddenness both of the onset of, and recovery from, unconsciousness in "faints" of epileptic nature. A fit occurring during sleep is invariably epileptic.

Even bearing these points in mind, it is not always easy to differentiate fits which are epileptic from those of hysterical nature. What appears to be a definite hysterical seizure may have an epileptic commencement; in this way the diagnosis may be difficult, as the epileptic phenomena may be brief and largely obscured by the hysterical. On this account the presence of an aura should be ascertained, and the exact mode of commencement of a convulsive seizure. Moreover, the existence of hysteria following upon an epileptic attack is not confined to the female sex, many male epileptics showing post-epileptic hysterical symptoms. The following features may be stated to be characteristic of an hysterical attack in contradistinction to epilepsy:—

1. Tongue-biting never occurs, but other parts of the body may be bitten as well as onlookers; involuntary micturition is unknown.

2. General struggling, throwing about of the body, kicking, fighting, etc., instead of the regular tonic, followed by clonic convulsion, of the epileptic.

3. The duration of the hysterical seizure may extend from a few minutes to a much longer time; the epileptic fit never exceeds a few minutes.

4. The epileptic fit terminates in deep sleep, the hysterical seizure at most in a dazed condition.

Reference has previously been made to the difficulty of distinguishing *le petit mal* with slight convulsion from fits due to organic brain

disease, in both instances the fit having a local commencement. Loss of consciousness, however, marks the attack as an epileptic, as distinguished from a Jacksonian fit. Should unconsciousness be present in fits arising from cortical irritative disease, other evidence of intracranial new growth will probably be found.

Most cases of infantile hemiplegia develop epileptic fits at some period of their existence. These attacks differ in no way from those of idiopathic epilepsy except in their local commencement, and in the fact that of the two sides the paralysed is the more extensively convulsed.

A form of epileptic seizure characterised chiefly by *vertigo* may be difficult to recognise as of this nature. If evidence of gross intracranial disease is wanting, or if there is no labyrinthine condition likely to give rise to it, the following points would indicate its epileptic character:—There is a periodicity about its onset, it is rarely accompanied by vomiting, such as characterises auditory vertigo; there is some, though slight, obscuration of consciousness, and it is amenable to treatment by the bromides.

An epileptic fit characteristic in all its features occurring in an otherwise healthy adult person is highly suggestive of intracranial tumour.

Prognosis.—Death during a fit, and as the direct sequence of the convulsion, is rare. Even when the fit is at its worst, and asphyxia seems imminent, recovery takes place. The epileptic may, however, be killed as an indirect result; thus he may fall and fracture the base of the skull, or he may tumble into water or into the fire. On the other hand, he may be suffocated by turning on to his face as a result of the convulsion.

The question is often asked, Is there a cure of epilepsy? There is no doubt that the prolonged and judicious use of the bromides may keep down the seizures, and in several cases arrest them completely; but it would be hazardous to say that in the latter instance the disease was cured. A good rule to apply in all cases in which bromide is well borne and of benefit is to continue its use for two years after the last attack, and even then it should not be suddenly stopped. It is also clear to those who had observed epileptics under the favourable circumstances of an epileptic colony (*vide* "Colonies," p. 161), that change of environment may prove of the greatest possible service and even arrest the fits; but it is by no means certain that on the discharge of the patient from the colony after two or more years' freedom from attacks there may not be a recurrence under the less favourable conditions of daily work and making a living.

The majority of epileptics show in process of time some mental deterioration (*see* "Epileptic Insanity"). The most favourable cases are those arising from scarlet fever without renal complications. Night fits are less influenced by treatment than those occurring only by day, and attacks of *le petit mal* are less susceptible to drugs than the severer forms.

Medicinal Treatment.—Since Locock, in 1857, introduced bromide of potassium in the treatment of epilepsy, this remedy and the salts of bromine with the other alkaline earths have been universally used to diminish the frequency and severity of the attacks as well as prevent their recurrence. Bromide of potassium is the remedy most commonly employed, but a combination of the bromides of potassium, sodium, and ammonium is of value in many cases. By far the largest number of cases do well under these salts, only in quite a minority they have no obvious influence upon the disease. The administration of the bromides should be continued over a long period of time. In the cases in which the drug is of value, there is nothing more common than to see a recrudescence of the fits at any break in its administration. In addition to the effect of bromide upon the fits, it is also of influence in clearing up the associated mental condition. Bromide may be administered in several ways, either in doses of 10 to 20 grains three times a day, or half-drachm doses night and morning; a large dose, 40 to 60 grains, given at bed-time is especially recommended in the case of nocturnal and matutinal fits. Bromide is best administered about one hour after a meal. Children bear the drug well.

If the onset of the fits is definitely periodic, or if a warning is given some time before its actual occurrence, a dose of bromide may often arrest or postpone the attack.

"Bromism" may arise from the prolonged administration of bromides. This is a condition in which the patient becomes dull and lethargic, the sensibility blunted, the memory and intelligence impaired, his extremities cold, the speech slow and saliva often runs from the mouth.

The use of bromide of potassium sometimes leads to an eruption of acne, a condition which is benefited by the salts of arsenic (*see* "Bromism").

Bromide is partly eliminated through the milk secretion, as "bromism" has been observed in children suckled by epileptic mothers under treatment by this drug. Bromide exhibited during pregnancy has no deleterious influence upon the fœtus. Even in the most favourable cases it is not wise to stop its use until two years have elapsed after the last fit: it may then be diminished to a dose once or twice a week, the interval between the doses being gradually prolonged.

Bromide may be satisfactorily combined with some other drug in special cases. In those with weak cardiac action the combination with tincture of digitalis is of service, while aconite or chloral may be judiciously given in those associated with a full and bounding pulse. In the young and anæmic it should be given along

with cod-liver oil or the hypophosphites and iron.

It is doubtful whether all the bromide salts are equally efficacious. Bromide of potassium appears to be the most valuable; after it come those of sodium and ammonium. Bromide of strontium, bromide of nickel, and bromide of camphor have a decidedly less potent action than the alkaline bromides. A combination of bromide and opium has been suggested, but it has no special therapeutic advantage.

In a few cases the bromides appear to exert no influence, hence it is necessary to rely upon other but less efficacious remedies. Perhaps the most valuable is borax, as recommended by Gowers. This may be given in doses of 10 to 20 grains thrice daily alone or in combination with bromide. Numerous other drugs have from time to time been tried, but none of them possess the specific action of the bromides. Such are belladonna, cannabis indica, the zinc salts, nitro-glycerine, salicylate of soda, antipyrin, sulphonal, and a host of other remedies. The use of nitrate of silver, a drug largely used before the introduction of the bromides, has been almost entirely discarded.

Errors of refraction are not uncommon amongst epileptics, but it cannot be said that the correction of astigmatism has ever cured epileptic fits. Such errors should be corrected when possible, but the medicinal and general hygienic treatment of the disease should be continued along the lines already laid down.

A word, in conclusion, may be said upon the diet of epileptics. If an epileptic has to go about and do the work of the average healthy individual he requires the ordinary mixed diet of such persons. The vegetarian *régime* recommended by some has not been found to possess that minimising influence upon the number and severity of the seizures which its advocates predicted. A salutary rule is to allow butcher-meat once daily and to interdict the use of alcohol, except upon emergencies.[1]

Epileptic Colonies.

See also Epilepsy.

The method of dealing with epileptics in colonies does not date farther back than the first half of the present century; for it was only in 1848 that John Bost, a Protestant clergyman at La Force, near Bordeaux, founded his homes for the treatment of the feeble-minded, idiots and epileptics. Since that time the value of the principle of epileptic colonies, the conception of which would seem to be due to J. Bost, has taken extensive hold upon those whose care or duty it is to deal with numbers of epileptics. It is only necessary to refer to the large and prosperous colony at Bielefeld in Westphalia, founded and extended through the influence and energies of Pastor Bodelschwingh, and to numerous others in various parts of Germany, which have arisen during the course of the past half-century. To mention the names of a few: there are those at Potsdam, Dalldorf,—Berlin, Stettin, Hubertusburg, and many others in Germany; those at Rolle, Zurich, and Berne in Switzerland; that at Haarlem in Holland; and the "asiles" John Bost and de la Teppe in France; the Ohio institution, the Craig Colony in New York State, and many others springing up in other North American States; and in our own country, Maghull, near Liverpool, the Meath Home at Godalming, the colony for epileptics at Chalfont St. Peter, Bucks, which is the property of the National Society for the Employment of Epileptics, and the colony founded by the David Lewis Trust at Chelford in Cheshire.

It is necessary at the outset to maintain that the general principle guiding the formation of colonies for epileptics should be the care, management, and treatment of the sane as contrasted with the insane epileptic, for whom provision is already made in the lunatic asylums. But it is well known that, owing to the close relationship between epilepsy, idiocy, and imbecility in early life and with insanity (dementia) in the later years of an epileptic's existence, it is not always possible to draw this strict line of demarcation. There are, however, numerous epileptics, seen chiefly in the out-patient rooms of the hospitals and in the wards of the Poor Law infirmaries, who, if suitable opportunities were afforded them, would be capable of carrying on a considerable amount of work towards their own maintenance. On the other hand, owing to deficient training in youth, or to inability to obtain regular employment on account of fits, or from the fact that the epileptic is excluded from many sources of employment, such as building, engineering, railway work, and so on, the services of the epileptic artisan are not sought after by the employer of labour. Hence not only amongst the working classes, but also amongst those in easier circumstances, the epileptic is found to be a burden to his relatives. Founded, therefore, upon such facts as these, quite apart from statistical proofs of their existence, attempts are being made by various institutions, charitable and other, to endeavour to do something to assist this sorely-afflicted class.

The problem is in process of solution, as already shown by the enormous and rapid growth

[1] See observations on diet in "Adolescent Insanity," vol. i. p. 59.

of the colony system. Into these institutions are taken epileptic children with a view to an education suitable to the peculiarities of their affliction, and epileptic youths and adults, with the object of affording them employment of a kind especially favourable to their disease; by which means it is hoped to ward off or delay the downward tendency towards dementia which is characteristic of most cases of severe or prolonged epilepsy.

Plan of an Epileptic Colony.—A properly designed epileptic colony should consist of various departments, each under the supervision of a competent head, the whole administrative control being under the care of a fully-qualified medical man. For not only have the inmates to be governed and educated, and have work apportioned to them according to their mental and physical capabilities, but they are sufferers from a disease having special peculiarities and characters which it is the business of a medical man to study and control. The colony should consist of:—

1. Residential buildings of the villa type, each to hold from 18 to 24 or 30 inmates. Each building should be complete in itself in so far as all domestic and sanitary matters are concerned. In charge of each should be a competent housekeeper. The houses for the males should be entirely distinct from those for females, and preferably at some distance from each other.

2. Farm, market garden, farm buildings, etc., and the bailiff's house. There is no form of outdoor work upon a farm or garden from which an epileptic may be debarred.

3. Workshops, such as carpenter's shop, the smithy, bootmaker's, tailor's, laundry, etc.

4. The schools, which should be attached to the residences of the children.

5. The sick-house.—This is an essential element of an epileptic colony. Many epileptics become patients after their attacks, and require careful nursing and attention, and in some cases restraint. Hence the possession of a padded-room is an important feature in the sick-house arrangements.

6. An infirmary for the old and demented.—It would appear as if this became a necessity in a colony as time goes on. Those in England have not been sufficiently long established for this necessity to have arisen; but in the older colonies on the Continent it has been found beneficial to transfer the aged and infirm to such a building rather than to an asylum.

7. The administrative department, consisting of the residence of the Superintendent and Matron, the general offices, and other necessary departments.

The Financing of an Epileptic Colony.—There are several ways by which money may be raised to originate, support, and maintain these institutions.

The first place is given to charitable contributions. Although money may be obtained from the sources to be immediately mentioned, it is necessary that this should form an important element in the financing of a young and developing colony.

2. By payments from the School authorities. —The education of the epileptic child falls as much under the care of the educational authorities as that of normal individuals. It is advisable, however, that in the majority of cases the epileptic should be educated apart from the non-epileptic, although there are individual instances which may be excluded from this general statement.

It has been suggested, and arrangements are likely to be made for the purpose, that the schooling of epileptic children of the poorer classes should be carried out in colonies for epileptics. From the purely medical side the following conditions appear to favour the separation of epileptic from healthy children. (*a*) Great frequency or severity of the seizures, even though there be no marked impairment of intellect. (*b*) If along with epilepsy there exists feeble-mindedness, but without actual idiocy or imbecility.

If there be no intellectual blunting, and the fits occur at long intervals or only at night, there is no indication to education apart from other children.

3. By payments from Boards of Guardians.—Owing to the detention in workhouse infirmaries of numbers of epileptics, many of whom are sane, able-bodied, and capable of work under direction, the guardians should have the power to pay for and maintain such persons in epileptic colonies, where their services may be usefully employed, and their disease treated upon physiological lines, so that they may be placed under the most favourable conditions for the amelioration of their disease and possible recovery.

But as a matter of much importance, it should be in the power of Boards of Guardians to assist the relatives to maintain an epileptic member of their family at one of the colonies without at the same time pauperising such individuals.

4. By the payment of colonists.—This is an important item, and a necessary one in the early stages of an unendowed colony. In some institutions three grades of colonists are admitted: the first, a highly-paying class, pay sufficient to cover their own expenses as well as to assist those of the third class, who are most likely unable to meet the full outlay required for their keep. The second or intermediate class merely cover their expenses by their payments.

In course of time the industrial or farming operations ought, under judicious care and management, to cover their own expenses, and perhaps afford a small balance of profit; but in a colony in full working order, with the necessary superintendent, heads of departments,

instructors, nurses, attendants, and servants, it is scarcely likely that the whole can be maintained without financial assistance from outside.

Influence of the Colony System upon Epileptics.—The remarks which follow are based on observations upon the epileptics at the colony belonging to the National Society for the Employment of Epileptics at the farm at Chalfont St. Peter, Bucks.

The guiding principles of the management of epileptics in colonies are :—

(1) Removal of the epileptic from the town to the country.—The observations which have been made on this point during the colony's existence amply testify to the good which this change is capable of producing. Many of the colonists arrive in a state of mental and physical debility out of all proportion to the frequency or severity of the epileptic seizures. Under these conditions it is impossible to put them upon full work. In a short time, however, aided no doubt by other ameliorating circumstances, the colonist is able to do his share, and to be the better of doing it.

(2) Regular employment.—This is a factor of the utmost importance, more especially when it is borne in mind that inability to obtain work, which is the common history before admission, has a bad influence upon the general condition of the epileptic. The epileptic is, as a rule, of a hopeful nature, and unless he is reduced by the disease to a state of permanent dementia, believes himself capable of effecting good and useful work. Encouragement in this direction is one of the objects of the colony system. Acting upon various observations which have from time to time been made, it is attempted to give each colonist that kind of work to which he most readily adapts himself, without at the same time confining him either to outdoor or indoor employment.

(3 and 4) A well-ordered and regular mode of life, with avoidance of excitement and abstinence from alcoholic liquors, and abundance of nourishment of a simple nature, are points which do not require more than to be mentioned.

I may now briefly consider the effect of such general care and treatment upon the individual epileptics, and for this purpose look at it from the following points of view :—

1. *Frequency of the Fits.*—In the majority a marked diminution in the number of the attacks is observed after admission to the colony. This is probably due directly to the removal of the epileptic from bad hygienic surroundings in the town to a pure country air, with abundance of simple diet and a well-regulated *régime.* In a minority there is a temporary increase in the number of fits, which may be due to the fact that previously to admission larger doses of bromide have been taken, this being entirely withdrawn except for a small dose at bedtime. In both instances after a short time the average number of fits is struck, and this is found to vary largely in different cases. The greater number of fits occur during the day, and by far the commonest time is during the first hour after rising in the morning. The attendants have found that fits are rare on going to bed, and that as a rule the patients are much better and brighter during the evening. A point of some interest is that more fits occur in the house than outside in the garden or fields, and this apart from greater difficulty in registering the number outside.

2. *Severity of the Fits.*—Here, again, there is a majority in which an amelioration in the severity of the attacks occurs, while in a minority no material alteration is noticed, and in some a decided tendency towards increase in severity. Upon this point, however, it is more difficult to obtain accurate observations. In some of the colonists severe fits gave place to attacks of *petit mal*; in others, attacks of an average nature became more severe.

3. *General Mental State.*—In quite a minority there has been a progressive tendency towards dementia. In some it appeared to be the natural course of the disease. In others it was owing to the frequency and severity of the fits, or of both combined. In the greater number no such mental deterioration has been observed. On the whole, the mental state of the colonists, considering the duration of the disease, is good. Some of them are capable of work requiring individual alertness and tact, while most are able to do good work under supervision. But the greatest benefit which this system of treatment is capable of producing lies in the general moral effect which it has upon the individuals. These persons amongst the poorer classes are in many instances regarded as "family lepers," the outcasts of the community, between whom and their relatives there is little sympathy. In a colony each epileptic is readily brought to see that he is as useful a person as his more highly favoured brother if placed under proper conditions. Here interest is taken in his work, in his mode of life, and in his amusements, with the natural result that he takes greater interest in himself, and obtains a feeling of higher self-respect.

4. *General Physical State.*—It is under this heading that the greatest improvement has been noticed. Without exception the general health of each colonist has materially improved. They all put on weight, and this notwithstanding the fact that they may be subject to frequent and even severe fits.

It is obvious that discretion should be used in the selection of applicants. The points to which attention is specially directed are :—Has the epileptic been unable to obtain employment owing to the frequency or severity of the seizures? Is he (or she) capable of work under direction? And it is clearly of importance to avoid selecting cases which show indications of

violence, mania, or other symptoms indicating insanity.

The general experience at the colony has proved that the younger the epileptic, and the shorter the interval between the onset of epilepsy and admission to the colony, the more favourable and satisfactory is the influence of this system upon the course of the disease. It would also appear as if female epileptics responded more favourably to the system than the male sex.

By way of conclusion, a summary of the evidence collected by the Charity Organisation Society from the results of this method of treating epileptics in the colonies on the Continent may be quoted :—"Any attempt to provide for the treatment of epilepsy, if large numbers have to be considered, should combine school-teaching with medical treatment and supervision. For young persons and adults regular but not too laborious employment is necessary. For all alike, for the furtherance of self-control, and for healthy enjoyment, a well-ordered home life is required, and these the colony system provides. Medically, if the serious nature of the disease be taken into account, the colony system produces the best results. For the worst cases, and to provide against the constant ailments of many of the colonists, hospital accommodation is necessary. This system will have to furnish accommodation suitable for patients of all classes, the highest, who may wish to have the best fare and rooms, and the lowest. The growth of the colony system is remarkable. It meets a very widely-felt need."

Epileptic Insanity.—Although epileptic insanity is a term justified by long usage and by the fact that it has certain well-defined features, it must be clearly understood that there is no group of mental symptoms pathognomonic of epilepsy. But if the more prominent ones are present and combined with certain facial characters, a strikingly complete clinical picture is presented, which the observer, having once seen, cannot fail to recognise as that of an epileptic imbecile or dement.

For *mental enfeeblement* may be said to be the predominating feature of insanity associated with epilepsy, and may be said to be further characterised by manifestations of general irritability and impulsiveness. Frequently with this is combined an exhibition of perverted frothy religiosity, a tendency to quote Scripture and hold improvised miniature services. A group of worshippers gathered around a garrulous, incoherent preacher in the exercising ground of an asylum may safely be diagnosed as epileptics. Next to cases of dipsomania they betray a degree of cunning and mendacity unequalled by any other class of insane patient, and in them more than any other class is the gregarious instinct shown. Towards their own affection they manifest a curious optimism—a refusal to confess that the fits really occur, or an ever ready belief that they have had their last fit, coupled with an entire disregard for any of the risks which their malady daily necessitates their running. If certain few and rare cases be excepted, in whom during the course of a lifetime occasional fits occur in such a sporadic fashion as to fail to produce any permanent mental clouding—indeed they have occurred in some of the most prominent *rôle* bearers of history—if these be excepted, it may be truly said that the goal of all epilepsies is towards mental enfeeblement. The character and depth of this enfeeblement largely depend upon the age of the patient at the time of its commencement. If such be before or during the developmental period the result is a condition of either imbecility or idiocy, according to the amount of normal mental development that had already taken place; if subsequent to that period, the resulting condition is that of dementia, the degree of which varies from simple blunting or clouding of the keener mental faculties on to illustrations of the most profound depths of dementia. The physiognomy, as in other varieties of mental disease, reflects this, but in addition often presents certain special characters; the skin frequently becomes coarse and persistently greasy, and is apt to assume a dirty hue or pallor, while the features may become thickened and ungainly, apart from the consequences of falls during the fits, which curiously so often result in repeated laceration of the same area; conjoined with this, and probably owning a common origin, the neck is prone to become thickened, giving it a short, bull-like appearance.

States of mental enfeeblement, however, by no means exhaust all the conditions under which epileptic insanity manifests itself. It is true they form the main underlying stratum, and may be of sufficient depth themselves to bring the patient under asylum control; but far more frequently a case of epilepsy is certified as insane as the result of the appearance of certain other mental symptoms, which may be regarded as adventitious. Such may be in the direction of depression, and constitute *epileptic melancholia* with or without suicidal proclivities; or the reductions may be deeper and result in various *maniacal outbursts*, sometimes slight, sometimes so grave as to include the severest of all forms of delirium and frenzy, with homicidal attempts. These phases may or may not be accompanied by *hallucinations*, often of a most distressing character, and which may affect one or all the senses; in addition to those of hearing and sight which are the commonest, formications are especially frequent among the epileptic insane. It is among them, too, that the best examples of *illusions* may perhaps be found, which fact may possibly explain their tendency to make the most barefaced, unfounded charges. Associated with the above is the development of numerous *delusions*, usually in the direction of suspicion

and persecution, not systematised, but more or less fleeting and changeable, and it is frequently upon these as a basis that many of the insane impulses of epileptics, often of a most terrible nature, are actuated. Such acts may, however, be examples of epileptic automatism. *Stupor* of the anergic variety is another very common phase, often broken in upon by brief intermissions of motor restlessness and jactitation. These adventitious mental symptoms may precede or, as is more customary, follow a fit or series of fits, or may occur in both these relationships, or, again, may appear to take the place of and represent the convulsions. The last relationship has been doubted, but it is certain that in some instances where the occurrence of fits shows a definite periodicity, an outcrop of mental symptoms occasionally occurs and apparently replaces a series of fits which, by experience of the case, those in attendance had learned to then expect; this manifestation has been dignified by the appellation of a special term—*épilepsie larvée.*

In by far the majority of cases the epileptic neurosis commences before the completion of the developmental period (25), but there are certain rare instances where, after the insanity has existed may years and dementia has supervened, the patient begins to have typical and regularly recurring epileptic fits. Another rare relationship of epilepsy to insanity is in a variety of melancholia (convulsive), in the course of which a few, perhaps three or four, fits occur in a patient who has passed the developmental period and was not previously the subject of epilepsy (Clouston). Nor must mention be omitted of cases of alcoholic insanity in middle life who develop fits, which recur at longer or shorter intervals, and in whom, contrary to the previous variety, the prognosis is hopeful.

The epileptic insane do not live long lives, and a large proportion either succumb to or die with pulmonary tuberculosis.

The *diagnosis* of epileptic insanity is not as a rule difficult. Certain cases sent in to an asylum as epileptics subsequently prove themselves to be examples of general paralysis,—a mistake readily made in the absence of adequate history and the presence, perhaps, of only a few faint tremors and a doubtful pupillary abnormality to suggest the latter malady. The diagnosis from hysteroid attacks is referred to in the article on "Epilepsy." Acts, especially of violence and crime, committed, as far as can be ascertained, in a state of false consciousness by a person who hitherto had been free from fits, present great difficulty, and, when involving medico-legal aspects as they so often do, the medical expert requires some courage to definitely diagnose them as manifestations of masked epilepsy (*épilepsie larvée*).

The *treatment* of epileptic insanity does not medicinally differ from that described above under "Epilepsy." The bromide of potassium alone, or combined with those of ammonium and sodium, or, of more recent advocacy, the bromide of strontium, may be with advantage administered in doses increased up to thirty or forty grains thrice daily, immediately after food, and, to yield their full benefit, should be given regularly over long periods, indeed they should be used as an article of diet. They diminish both the number and severity of the fits, and have a salutary effect upon the frequency and character of the mental phenomena. Where maniacal excitement is continued over long intervals, Indian hemp in half-drachm doses of the tincture should be added. For more urgent use, such as in the control of furious maniacal outbursts, chloral is invaluable, especially when the patient is separated from the ward to the quietude of a single room. This drug is also our mainstay in the cutting short of a *status epilepticus*; in this condition it should be administered *per rectum*, and one large dose (40 to 60 grains) is less dangerous than several repeated moderate doses. Its cardio-depressant action may be counteracted by the conjoined administration of brandy, with perhaps a subcutaneous injection of atropine. As to food, where the dietary is generous, the use of animal food should be limited, but it must be sufficient to replace tissue waste where resort is had to much out-door manual labour, —a most valuable mode of treatment, and one for which colonies are gradually coming into vogue. Strict attention should be paid to the bowels and excretory functions in general; indeed, many hold that certain cases of epilepsy with or without mental phenomena are autotoxic from an overladen gastro-intestinal tract, or one whose chemistry is faulty. Much tact and moral persuasion is needed in the successful management of a ward where a number of epileptics are together; listen and return a non-controversial answer is a good axiom. Their gregarious instinct may be profitably utilised, for there is no class of patient so ready to succour a similarly afflicted comrade. Their tendency to quarrelsomeness and pugnacity may be minimised by an ample allowance of floor-area; in other words, epileptics require elbow-room. The recovery rate among this class of patient is considerably less than among the remainder of a mixed asylum population, but is sufficiently large to encourage every possible effort of those in charge of them.

Epilepticism.—The status epilepticus. *See* Epilepsy.

Epimanes.—The raving stage in insanity; a patient in a paroxysm of insanity.

Epinephrin.—An astringent and hæmostatic powder prepared from the suprarenal capsule. *See* Adrenal Glands, Adrenalin; Anæsthetics (*Other Anæsthetics*); Hemisine.

Epinephritis.—Inflammation of the suprarenal gland. *See* ADRENAL GLANDS (*Other Diseases*).

Epinyctis.—A skin disease characterised by the nocturnal eruption of pustules, accompanied by pain.

Epiphenomenon.—A secondary symptom; one superadded to the more essential manifestation of the malady.

Epiphora.—"Watery eye"; an overflow of tears due to over-secretion or to some fault in the lachrymal apparatus (blocking of the nasal duct, narrowing of the puncta, etc.). *See* EYE, CLINICAL EXAMINATION (*Symptomatology*); LACRIMAL APPARATUS, DISEASES (*Excretory Apparatus, Puncta and Canaliculi*); NOSE, NASAL NEUROSES (*Hydrorrhœa*); SYPHILIS (*Tertiary Symptoms, Eye and Appendages*).

Epiphyses. *See also* FRACTURES (*Separation of Epiphyses*); HIP-JOINT, INJURIES OF (*Epiphysial Separations*); KNEE-JOINT, INJURIES (*Epiphysial Injuries*); SHOULDER, DISEASES AND INJURIES (*Separated Epiphyses*); SYPHILIS (*in Children, Bone Changes*); PHYSIOLOGY, TISSUES (*Bone*).—The injuries and diseases of the individual epiphyses will be referred to in the articles dealing with the different segments of the skeleton, *e.g.* shoulder, elbow, hip, etc.; the present article is concerned with certain general considerations. Any account of the surgery of the epiphyses must refer, not only to the independent centres of ossification known as epiphyses, and the disc of cartilage by means of which they are united to the shaft, but also to the zone of active ossification on the diaphysial aspect of the disc, known as the ossifying junction or bulb of the bone. It is by involvement of the latter that injuries and diseases of the epiphyses may seriously interfere with the growth of the bone concerned; this interference will vary with the nature and extent of the lesion, with the physiological importance of the epiphysial junction, and with the amount of work which remains to be done before the skeleton attains maturity, or, in other words, with the age at which the lesion occurs.

The variation in the physiological importance of epiphysial junctions may be illustrated by referring to those at the knee, which contribute a great deal more to the length of the lower limb than do those at the hip or ankle, while the former are the last to unite with their respective shafts; in the upper limb this arrangement is reversed, for the more actively functionating epiphysial junctions are met with at the shoulder and wrist, which, like those at the knee, are also the last to unite. Again, the results of interference with growth will be more injurious in the lower than in the upper limb, because, from the functional point of view, it is essential that the lower extremities should be, approximately, of equal length. Similarly, the interference with growth of one of two parallel bones, *e.g.* in the forearm or leg, is most undesirable, for if the growth of one of them is arrested while that of the other continues, there results a deviation of the hand or foot to one or other side. It occasionally happens that the interference with the ossifying junction results in increased activity of growth, and increase in length of the bone concerned.

It is also important to bear in mind the anatomical relationships of any epiphysis to the adjacent joint, for the latter may be implicated by any injury or disease affecting the former; the part played by injury in this relation is well illustrated at the elbow, where epiphysial separation at the lower end of the humerus may seriously impair the future usefulness of the joint; the best illustration of disease in an epiphysis or at the junction with the shaft affecting the adjacent joint is found at the upper end of the femur, where the epiphysial junction is entirely intra-articular. The structural relationships of the epiphyses are such as render them liable to certain special forms of injury which, in patients under twenty-one years of age, take the place of fractures of the joint ends of bones and of the dislocations of adult life.

The arrangement of the blood-vessels in the epiphyses and at the ossifying junctions is such as favours the development of bacterial diseases, and the complicated nature of the histological changes at the epiphysial junctions during the period of growth probably plays an important part in the liability of the tissues concerned to give rise to new growths of the bony, cartilaginous, and sarcomatous types.

INJURIES.—*Epiphysial sprains and incomplete separation of epiphyses* are common enough in infants, but owing to the absence of displacement they often escape notice, especially in mild cases; they are chiefly of importance in favouring the development and localisation of bacterial diseases at the time of the injury or at any subsequent period.

Complete separation of an epiphysis may take place through the epiphysial cartilaginous disc, or exactly at the epiphysial junction, so that the cartilaginous disc clings to the epiphysis, or through the ossifying junction, so that part of the young bone belonging to the shaft remains attached to the epiphysis. The last of these, which is also called *epiphysiary fracture*, is by far the most common. The displacement of the separated epiphysis may be complete or incomplete; it largely depends on whether the periosteum is or is not torn, and on the degree and nature of the violence. Dislocation of the epiphysis from its articular relations is exceedingly rare.

Epiphysial separations may be simple or

compound; in the latter the end of the shaft is usually stripped of periosteum, and protrudes through the skin. Separation of the epiphysis may be complicated with partial, often splintered fracture of the adjacent shaft, or with fracture of the epiphysis itself, with injury to blood-vessels, nerves, muscles, tendons; separation of the lower femoral epiphysis is a good example of one attended with injury to the adjacent (popliteal) vessels and nerves.

The violence causing separation of epiphysis is usually indirect, such as falling on the feet or hands from a height, entanglement of the limb in the wheel of a vehicle, violent traction on the limb, attempting the reduction of deformities, *e.g.* that left by tubercular disease of the knee. They may also result from direct violence. The frequency as to age varies with the individual epiphysis, but the majority are met with between the ages of 11 or 12 and 18 years. The adjoining table of the times of union of the different epiphyses is copied from Poland's valuable work:—

Upper epiphysis of radius	At the 16th year.
Olecranon epiphysis of ulna	Between 16th and 17th years.
The lower epiphysis of humerus	At the 17th year.
The internal epicondyle of humerus	At the 18th year.
Phalanges of toes	Between 17th and 20th years.
Phalanges of fingers	Between 18th and 20th years.
Lower epiphysis of ulna	Between 18th and 20th years.
Metacarpals	About the 20th year.
Upper epiphysis of humerus	Between 18th and 22nd years.
Lower epiphysis of radius	Between 19th and 23rd years.
Epiphysis of clavicle	Between 22nd and 25th years.
Lesser trochanter	At the 18th year.
Great trochanter	Between 18th and 19th years.
Metatarsals	Between 19th and 20th years.
Head of femur	At the 19th year.
Lower epiphysis of tibia	Between 18th and 19th years.
Lower epiphysis of fibula	Between 19th and 21st years.
Upper epiphysis of fibula	Between 20th and 22nd years.
Upper epiphysis of tibia	Between 21st and 22nd years.
Lower epiphysis of femur	Between 20th and 23rd years.

The order of frequency obtained from recorded cases is as follows:—(1) The lower femoral, (2) the lower radial, (3) the upper humeral, (4) the lower humeral, (5) the lower tibial, (6) the upper tibial epiphysis. The male sex shows an enormous preponderance, doubtless from the greater exposure to injury between the ages of 11 and 18, during which period the majority of separations occur.

The repair of the injury is usually quite satisfactory; non-union is extremely rare; deformity from vicious union, exuberant callus interfering with an adjacent joint, and interference with the subsequent growth of the bone involved, are exceptional.

Pathological or spontaneous separation of epiphyses, like spontaneous fractures, are the result of *diseased conditions*, which in the case of the former are chiefly *localised at the epiphysial junctions*; they include *rickets*, *scurvy*, *inherited syphilis*, *suppuration*, *tuberculosis*, and *sarcoma*, and need not be further referred to in this place, as they are described under separate heads (Bones, Joints, and the Individual Joints).

Epiphysiolysis.—Separation of an epiphysis from a long bone. *See* EPIPHYSES.

Epiphysitis.—Inflammation of an epiphysis. *See* BONE, DISEASES OF (*Pyogenic, Suppurative Osteo-myelitis*); BONE, DISEASES OF (*Syphilitic Diseases, Epiphysitis or Osteo-chondritis syphilitica*); HIP-JOINT, DISEASES OF (*Syphilitic*); PARALYSIS (*Acute Anterior Polio-myelitis, Diagnosis from Acute Epiphysitis*).

Epiphyte.—An ectoparasite of a vegetable nature. *See* SKIN, PARASITES; FAVUS; etc.

Epiplocele.—Hernia containing omentum. *See* HERNIA (*Structure, Contents*).

Epiploon.—The omentum.

Epiplopexy.—An operation in which the omentum is fixed to the abdominal wall; it has been employed in ascites due to hepatic cirrhosis.

Episcleritis—Inflammation of the subconjunctival connective tissue. *See* SCLEROTIC, DISEASES OF (*Episcleritis*); GOUT (*Irregular, Eye*).

Episiocele.—Hernia of the vulva or pudenda.

Episiocleisis.—Closure of the vulva by the setting up of labial adhesions, *e.g.* in incurable cases of vesico-vaginal fistula.

Episiorrhaphy and **Episio-perineorrhaphy.**—Plastic operations for the repair of the vulva or of the vulva and perineum in cases of prolapsus uteri. *See* PELVIS, PERINEUM AND PELVIC FLOOR (*Prolapsus Uteri, Treatment*).

Episiotomy.—The making of an incision (unilateral) or of incisions (bilateral) in the vulvar margin in order to allow the birth of the head in labour and to prevent rupture of the perineum in the middle line. *See* LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Injuries to the Perineum, Prevention*).

Epispadias.—An open condition of the urethral canal, the upper wall being defective and there being a fissure in the dorsum penis; a similar condition may be found in the female urethra, but much more limited in its extent on account of the shortness of the urethra in women; this malformation is often associated with extroversion of the bladder. *See* BLADDER, INJURIES AND DISEASES OF (*Malformations*); HERMAPHRODITISM; SCROTUM AND TESTICLE, DISEASES OF (*Sterility*); URETHRA, DISEASES OF (*Abnormalities, Epispadias*); UTERUS, MALFORMATIONS OF.

Epispastics.—Substances which when applied to the skin cause blisters; vesicants (Gr. ἐπί, outwards, and σπάειν, to draw). *See* CANTHARIDES; PHARMACOLOGY; PRESCRIBING.

Epistaxis.

See also Glanders (*Symptoms in Man*); Hæmatemesis (*Conditions which simulate*); Hæmoptysis (*Diagnosis*); Hysteria (*Symptoms, Circulatory Disorders*); Leprosy (*Clinical Features*); Leucocythæmia (*Symptoms*); Liver, Diseases of (*Hypertrophic Biliary Cirrhosis, Symptoms*); Liver, Diseases of (*Signs of Cirrhosis*); Mumps or Epidemic Parotitis (*Complications*); Nephritis (*Renal Cirrhosis*); Purpura (*Werlhof's Purpura, Symptoms*); Raynaud's Disease (*Progress and Complications*); Scurvy, Infantile (*Clinical Features*); Typhoid Fever (*Symptoms*); Undulant Fever (*Symptoms*).

Definition.—The escape of blood from the mucous membrane of the nose.

Frequency.—Pliny expresses the opinion that "man is the only creature from whom blood flows at the nostrils." Only excepting menstruation, there is no hæmorrhage of greater frequency and none regarded with less concern by the public mind. Occasionally it causes anxiety, more often it is welcomed, and generally the bleeding is forgotten as soon as it has ceased. For this latter reason it is difficult to obtain very accurate statistics as to the frequency of epistaxis.

From a consideration of a large mass of statistics based upon questions put to many hundreds of patients of both sexes, of all ages, and of all conditions of health, the writer found that one hundred and forty-two per thousand had recollections of blood dropping from the nose; and that it was of rather more frequent occurrence in the male sex.

Age.—The periods of life during which it is most prone to occur were found to be between the ages of 10 and 25, and after 55.

Pathogenesis.—Epistaxis occurs in one of two ways. Blood may escape from the veins and capillaries by *diapedesis*, in which case the loss of blood is gradual, and although not great at any one time, yet if the diapedesis is long continued or often repeated, in the aggregate the loss may be serious. The other way in which epistaxis may occur is by *rupture of a blood-vessel*; the onset is then more sudden and more copious, but the total amount of blood lost is not necessarily greater than by diapedesis. In this latter way only can the bleeding be arterial.

Epistaxis by diapedesis implies increased blood-pressure in the veins and capillaries, or increased permeability of the walls of the vessels. The increased permeability of the vessel wall may be due to defective nutrition (blood diseases); to the introduction of certain poisons or poisonous products into the blood (infectious fevers, toxæmic affections, phosphorous poisoning); or to a defect in the vessel wall which may be congenital, as in the case of so-called "bleeders" (hæmophilia), or acquired, as in purpura or scurvy.

Epistaxis through rupture of a vessel is usually traumatic, rupture may result from increased blood-pressure in senile and degenerate vessels, or vessels may be laid open by ulceration and new growths.

The *bleeding point* in the vast majority of cases, and certainly of those due to constitutional causes, is to be found in the anterior and lower part of the cartilaginous septum. At this spot there is a very free anastomosis of the terminal twigs of the vessels supplying the septum. Other parts of the nasal chambers in the lower animals as well as in man, however, are equally rich in blood-vessels, so that vascularity in itself does not serve to adequately explain the proclivity of the blood-vessels at this spot in man to allow blood to escape so often, and apparently with so little provocation. A consideration of the development of the nose led the writer to offer the following as a further explanation. The structures entering into the formation of the septum are subject to delayed or excessive development, and their developmental activity continues not only up to puberty, but in a greater or less degree for some years after, as evidenced by the formation of ridges and spurs. In consequence the blood-supply to this region is carried partly by newly-formed vessels, these are more easily torn and more readily permit diapedesis to take place.

The human face in comparison with that of the lower animals is characterised by being extended vertically instead of horizontally. This characteristic is determined by the development of the septum. In the foregoing remarks we have a corollary to the opinion expressed by Pliny, and quoted above, "that man is the only creature from whom blood flows from the nostrils."

Etiology.—Epistaxis may be *primary*, *i.e.* arising from some abnormal condition in the nose, and therefore local in origin; or *secondary*, *i.e.* arising from some abnormal constitutional condition.

Amongst the primary or local causes, excepting developmental, the most frequent is *traumatism*. The injury may be direct or indirect; it may be excited by the introduction of foreign bodies into the nostril, as is often a habit with children; by picking the nose, at times resorted to by those who have experienced relief from an epistaxis in order to artificially induce another; or by a blow directly upon the nose. When the blow is sufficient to cause fracture of the septum the hæmorrhage may be more than an epistaxis,

or it may be entirely a submucous hæmorrhage or hæmatoma. Amongst the indirect injuries fracture of the base of skull passing through the anterior or middle fossa may give rise to epistaxis.

All diseases of the nose, including nasal polypi, must be considered amongst the local causes. The separation of crusts which have formed upon the nasal mucous membrane may lead to erosion and ulceration from which epistaxis may occur. The ulceration produced by syphilis, lupus, leprosy, and tuberculosis seldom occasions epistaxis by a direct extension to the blood-vessels.

Of all local conditions *neoplasms* are more particularly liable to give rise to very violent attacks of hæmorrhage. Especially the fibromata, fibro-sarcomata, the angeio- and myxo-sarcomata, and the carcinomata.

The changes produced in the nasal mucosa by the presence of *bacteria*, as in nasal diphtheria, may be accompanied by considerable epistaxis.

Epistaxis occurring without any apparent local cause about the years of *puberty* calls for special attention, partly on account of its periodicity and frequency, and partly on account of the significance and etiology that has been attached to it. Those attacked are usually in normal health, some are plethoric and some anæmic. It has been described as *habitual*, as the epistaxis of *puberty*, and as *vicarious epistaxis*. There is evidence to show that some relationship exists between the generative function and epistaxis. Epistaxis undoubtedly may precede a menstrual flow, or accompany or follow one, or the menstrual flow may be entirely replaced by an epistaxis. On the other hand there is also evidence to show that other regions of the body are more frequently than the nose the seats of vicarious hæmorrhage. It is as well, therefore, to hesitate before too readily assigning a vicarious character to epistaxis. For reasons stated in discussing the pathogenesis the term *developmental epistaxis* would, perhaps, more completely cover this group of cases.

The constitutional conditions predisposing to epistaxis are *blood diseases* and those diseases giving rise to a *hæmorrhagic diathesis*, such as leukæmia, scurvy, purpura hæmorrhagica, hæmophilia. The *infectious fevers*, which may assume hæmorrhagic characters, at times are ushered in by an epistaxis; this is peculiarly so with typhoid. Conditions leading to *venous obstruction* or *increased arterial tension* are important factors; in illustration of the former mention may be made of tumours of the neck, whooping-cough, mitral stenosis, hepatic cirrhosis; and as illustrating the latter, hypertrophy of the left ventricle of the heart and renal disease.

Symptomatology.—Premonitory symptoms at times are experienced, a feeling of pressure and fulness within the head, frontal headache, dizziness, drowsiness, and tinnitus; these symptoms are met with in others besides those of plethoric habit.

The bleeding from the nostril in the majority of cases is trivial and in drops of a dark colour. If the bleeding is sufficiently profuse as to flow in a continuous stream it is probably arterial, occasioned by the rupture or ulceration of a small artery.

The blood at times, according to the position of the head and the profuseness of the bleeding, may pass backwards and over the septum, and escape from the other nostril, or it may pass into the throat and be coughed up, simulating an hæmoptysis.

The symptoms resulting from an epistaxis will be determined entirely by the amount of blood lost and the condition of the patient previous to the hæmorrhage. After a prolonged but gradual bleeding, anæmia may ensue; if the hæmorrhage is short and sudden but severe, syncope may occur, during which there is the possible danger of blood finding its way into the air-passages.

The local signs of a recent epistaxis are a sodden condition in the neighbourhood of the bleeding point, and crusts that have formed in the clotting.

Diagnosis.—It is impossible to lay too much stress upon the importance of looking for the bleeding point. In this way only is it possible to ascertain the real nature and significance of an epistaxis.

An anterior or a posterior rhinoscopy will generally enable one to decide whether the bleeding comes from either or both nostrils, or from the post-nasal space, and what steps should be taken for its arrest.

By the blood passing backwards into the pharynx, or being swallowed, a true epistaxis may simulate hæmoptysis or hæmatemesis. On the other hand, blood from the stomach or air-passages, by the act of vomiting or coughing, may be forced into the posterior nares and suggest an epistaxis.

It will often have to be decided by rhinoscopy whether the hæmorrhage arises in the nose or the post-nasal space.

Local Treatment.—There are times when an escape of blood from the nostrils is salutary, and the question then to be considered will be whether it is advisable to interfere. Having regard to the frequency of epistaxis the number of occasions surgical skill is called for to arrest it is comparatively small. In the majority of cases homely remedies are sufficient. Failing these we have a long array of astringent drugs and hæmostatics to fall back upon. The solutions to be effectual must be concentrated, and carried directly to the bleeding point; during the hæmorrhage this is seldom practicable. Moreover, there is the danger with concentrated

solutions of permanently injuring other parts of the nasal mucous membrane. After the hæmorrhage has ceased, however, hæmostatics are of service in sealing off a bleeding point and preventing a recurrence.

When the bleeding is due to an erosion or rupture of a small vessel on the septum a plug of cotton-wool inserted into the nostril together with digital pressure exercised from without, will generally bring about an arrest of the hæmorrhage. The lateral posture, with the head inclined slightly forward and the bleeding nostril uppermost, is the best. In this position blood flows less readily into the pharynx, and coagulation is more speedily obtained. The healing process can subsequently be accelerated by the application to the bleeding point of chromic acid fused on the end of a probe in consequence of which a reactive inflammation and subsequent cicatrisation is induced. A similar result may be obtained by gently stroking the part with the galvano-cautery, using a flat blade which should be kept at only a dull red heat.

When the hæmorrhage is more severe and the bleeding point farther back, and, perhaps, resulting from a surgical operation, the nostril can be packed. To do this one seldom has to resort to the use of any of the many surgical appliances ingeniously designed for the purpose. Packing is readily done with a strip of gauze from half an inch to an inch in width, impregnated with an antiseptic, and wrung out in an antiseptic solution. With the help of a nasal speculum the end of a strip is carried on a pair of nasal forceps as far back into the nostril as possible, the strip is then gradually and neatly packed away. The nostril is thus plugged from behind forwards. The strip should be removed after twenty-four hours, and the nostril, if necessary, repacked.

In the case of hæmorrhage persisting after a surgical operation, it is as well to syringe the nostril with cold water, and ascertain by inspection whether the bleeding is kept up by any tag of tissue not completely severed.

General Treatment.—In deciding upon the measures to be adopted to arrest an epistaxis it is important to bear in mind that the hæmorrhage is frequently but a local manifestation of a constitutional condition calling for treatment. The employment of appropriate general therapeutic means then becomes of the first importance.

Episympus.—A teratological type in which there is rotation outwards of the lower limbs and union of them by skin or membrane; the earliest stage in the production of sympodia. Vide *Antenatal Pathology* (vol. ii. p. 573).

Epithelioma. *See* Tumours (*Epithelial Group*). *See also* Bone, Diseases of (*Tumours*); Cicatrices (*Malignant Diseases of*); Kidney, Surgical Affections of (*Tumours*); Larynx, Malignant Disease of (*Pathology*); Mouth, Diseases of (*Diseases of Lips, Epithelioma; Diseases of Gums, Epithelioma*); Nose, Diseases of Nasal Orifices and Septum (*Malignant Growths of Nasal Fossæ*); Penis, Surgical Affections of (*New Growths, Malignant*); Scrotum and Testicle, Diseases of (*Epithelioma of Scrotum*); Tongue (*Tumours, Carcinoma, Epithelioma*); Tumours of the Skin (*Epithelioma adenoides cysticum*); Uterus, Malignant Tumours of; Vulva, Diseases of (*Tumours, Carcinoma*).

Epithelium. *See* Physiology, Tissues (*Epithelium*); Skin, Anatomy and Physiology (*Epidermis*); Skin, Pigmentary Affections (*Epithelial Pigmentation*).

Epithymia.—A natural desire, longing, or craving (Gr. ἐπιθυμέω, I set my heart upon).

Epitoxoid. — "Any toxoid which has less affinity for an antitoxin than the toxin has" (*Newman Dorland*).

Epitrichium.—The outer layer of the ectoderm in embryonic and early fœtal life; its outer cells are dome-shaped; it is supposed to persist in cases of fœtal ichthyosis or hyperkeratosis congenita. *See* Embryology (Fifth Week).

Epitrochlea.—The inner condyle of the humerus.

Epitympanum. — The attic of the internal ear.

Epityphlitis. — Appendicitis. *See* Appendix Vermiformis (*Appendicitis*).

Epizoa. — External animal parasites, as contrasted with *Epiphytes*, or external vegetable parasites. *See* Pediculosis; Scabies, etc.

Epizootic.—A term applied to diseases among animals which behave as epidemics do in the human subject.

Eponychium.—The thickened epitrichium at the end of a digit from which the nail is afterwards developed.

Epoophoron.—The organ of Rosenmüller or parovarium. *See* Broad Ligament (*Anatomy*); Generation, Female Organs of (*Organ of Rosenmüller*).

Epsom Salts.—Sulphate of Magnesium. *See* Constipation; Magnesium; Pharmacology; etc.

Epulis.—Any tumour growing from the gum or jaw, or, more strictly, a sarcomatous growth arising at or near the neck of a tooth. *See* MOUTH, DISEASES OF (*Diseases of the Gums*).

Equifex Disinfector.—An apparatus by which disinfection of articles is carried out by means of saturated steam.

Equilibrium.

See BRAIN, CEREBELLUM, AFFECTIONS OF (*Experimental Physiology, Equilibration*); PHYSIOLOGY, NERVOUS SYSTEM, CEREBELLUM (*Functions*); TABES DORSALIS (*Symptomatology, Disturbance of Gait*); VERTIGO.

PHYSIOLOGY.—The mechanism which is concerned in maintaining our bodies in a state of equilibrium, both during station and locomotion, and which provides for the due co-ordination of our various movements, is very complex, and our knowledge of the subject is meagre. There is a central apparatus, together with afferent and efferent channels, which convey impulses to and from it; and on the integrity of this machinery as a whole, and on the harmonious working of its component parts, the maintenance of equilibrium depends. The afferent channels are sensory nerves from the skin, muscles, and joints, special sense nerves, such as that of sight and hearing, and especially the vestibular portion of the auditory nerve which receives impressions from the ampullæ of the semicircular canals, and from the utricle and saccule, and visceral and other nerves, which convey obscure impulses from the viscera and other tissues. The precise paths in the spinal cord by which the afferent impulses reach the central apparatus, and what parts of the central nervous system are comprised in the central portion of the machinery, are all points that are very imperfectly known. The posterior columns of the cord are certainly concerned, as are probably the cerebellar tracts, while the cerebellum, corpora quadrigemina, pons, and medulla appear to be integral parts of the central apparatus, though the cerebrum is not wholly unconcerned in the matter. The motor paths and nerves supply the channels by which impulses are conveyed to the muscles concerned, which are principally those of the lower extremities and trunk. Although consciousness may be called into play, this is not essential, in that guiding impressions may reach the centres concerned with equilibration, and may generate impulses which determine muscular contractions for the co-ordination of our movements without our being distinctly conscious of this.

A simple procedure which serves to demonstrate the part played by impulses derived from cutaneous surfaces is that of rendering the soles of the feet anæsthetic by freezing the skin, for under such circumstances marked disturbance of equilibration becomes evident when an attempt is made to stand or walk. That other nerves, presumably from muscles and joints, are also concerned is made probable by the disturbances of co-ordination that may be met with in the absence of cutaneous anæsthesia, as in the subjects of a disease such as locomotor ataxy. The importance of impulses derived from the semicircular canals may be determined in various ways. Thus, if a person with eyes closed be placed absolutely immobile, in the recumbent position, on a horizontal table which can be made to revolve, his ability to say in which direction he has been moved, and in some people the degree of displacement that has taken place, is the result of knowledge derived from the semicircular canals, though it may be that vaso-motor and other obscure influences also play a part in this connection. Moreover, experimental destruction of the semicircular canals in animals results in profound disturbances of equilibration, a state of things that also obtains if the auditory nerve be divided or irritated.

It is not so easy to determine with certainty what centres are directly concerned with this function, in that the disturbances of equilibration which result from destructive lesions of certain parts of the central nervous system may equally well be due to the interruption of paths through these regions, by which impulses habitually pass to centres concerned with equilibration, but which are situated elsewhere, as by supposing that in the part destroyed are situated the centres concerned with the maintenance of equilibrium. Moreover, it is not easy to exclude the possibility that new and unusual impulses are generated at the seat of lesion, which on reaching the centres concerned with equilibration cause the disturbances which are seen in these cases. The most striking phenomenon that results from experimental ablation of parts, or of the whole of the cerebellum, is disordered equilibration, and the same obtains when any of the peduncles of the organ are divided. Section of one crus cerebri is attended with a like result, as are lesions of the corpora quadrigemina, pons, and medulla oblongata. It is of interest to note further that even lesions of the cerebrum have been attended by such disturbances in some instances. Moreover, as showing that the part played by the cerebrum in the maintenance of equilibrium is of considerable importance, may be instanced the fact that if ablation of the motor cortex be practised in a

dog that has previously recovered from the incoördination consequent on removal of the vestibule on both sides, the disturbances of co-ordination once more become manifest. So too it is found that the loss of ampullar influences is compensated for in a pigeon whose cerebral hemispheres are intact, but not in one in which they have been removed before the operation on the semicircular canals.

Disorders of Equilibration. — In clinical work we meet with a large number of affections, notably of the nervous system, in which disorders of equilibration occur as one of the manifestations that make up the symptom—complex, labelled as this or that disease. Moreover, as may be supposed from what has been said in connection with the anatomical and physiological parts of this article, diseases affecting very widely different parts of the nervous system may be attended by disturbance of equilibrium. Under such circumstances there may only be a subjective sensation of giddiness, or there may be objective staggering, while in a small number of cases forced movements occur in which the patient may, for instance, rotate about his longitudinal axis.

In multiple peripheral neuritis it sometimes happens that the chief motor disturbance is ataxy or incoördination, and so striking is this in some cases that the condition has been spoken of as pseudo-tabes or neuro-tabes, owing to the resemblance which cases of the kind bear to the disease tabes dorsalis, or locomotor ataxy, whose chief seat is the spinal cord, the posterior columns of which are sclerosed. In this latter affection, as one of its names implies, a prominent feature in the symptomatology of the majority of the cases is incoördination or ataxy, which may be manifested by an utter inability on the part of the patient to stand or walk. When the disturbance is less marked progression is possible, though the gait is ataxic; so too the patient may be able to stand, though on attempting to do so with the feet together and the eyes closed he sways, and if unsupported, tends to fall (Romberg's sign). In other words, what he is able to accomplish by the aid of sight is no longer possible when this afferent channel is closed, as well as those which transmit impulses from the skin, muscles, and joints of the lower extremities. In other diseases of the spinal cord in which the posterior columns are affected incoördination is a common feature. Friedreich's ataxy is an instance of this, as is cerebellar heredo-ataxy, although in these diseases the sclerosis of the cerebellar tracts may play a part in the causation of the disturbance of equilibration, while in the latter atrophy of the cerebellum may further contribute to this end. In the affection known as ataxic paraplegia, which is said to be due to a combined sclerosis of the posterior and lateral columns of the spinal cord, disturbances of equilibration are present to a variable degree, and unsteadiness may be one of the earliest symptoms of the important group of cases of acute combined degenerations of the spinal cord for which there has as yet been no very satisfactory name proposed. Disseminate sclerosis supplies an example of a disease in which disturbances of co-ordination may be due to interruption of afferent or efferent channels by the sclerotic patches, or of implication of some part of the central machinery which is concerned in maintaining equilibrium.

Tumours, hæmorrhagic extravasations, and softening from vascular occlusion, may each of them be responsible for disorders of equilibration if the lesion be situated in the medulla oblongata, pons, or corpora quadrigemina; but in none of these situations is the morbid process so liable to be attended by this symptom as when the cerebellum is affected, and, moreover, when the lesion is in the pons the most pronounced disturbance of equilibration results when the middle peduncle of the cerebellum is involved. Incoördination, the result of cerebellar disease, is commonly manifested by a reeling or staggering gait, which resembles that of an inebriated person; but sometimes there is a forced movement in some direction, *e.g.* rotation of the individual around the longitudinal axis of his body, or there may be a strong subjective sensation of such rotation even where no actual movement occurs. Forced movements of the kind have usually been associated with lesions of one or other of the peduncles of the cerebellum rather than of the organ itself.

Various affections of the brain are attended by disturbance of equilibrium, but here the subjective sensation of giddiness is more commonly met with than any objective evidence as manifested by disorders of station or locomotion. Thus it is that giddiness is one of the commonest auræ in epilepsy; so too in old people with degenerate arteries, whose brains are presumably ill-nourished, attacks of giddiness with, it may be, outward manifestations of this frequently result, or the giddiness may usher in an attack of cerebral hæmorrhage or thrombosis. Of tumours in the cerebral hemispheres those in the frontal lobes are especially liable to be attended by giddiness, and in some cases there is actual unsteadiness resembling that which may occur in cerebellar disease.

Erroneous visual perceptions consequent on paralytic defect of an ocular muscle cause disorders of equilibration, which may be recovered from, in spite of the ocular defect persisting, in that the centres concerned with the maintenance of equilibrium learn to disregard the erroneous impressions received. More striking and important are the disturbances of equilibration consequent on interference with the auditory nerve in so far as its vestibular portion is concerned. Tumours and the like, involving this nerve at the base of the brain,

may cause the disorder, but the most notable disturbances due to morbid processes in this sphere are seen in Menière's disease, otherwise known as auditory vertigo, in which the labyrinth is supposed to be the seat of the morbid change. The subjects of this malady are prone to attacks of vertigo, which may be very severe, and which may even cause the victim to fall with the suddenness of one shot. The nocturnal vertigo experienced by some people on first going to sleep has also been regarded as of labyrinthine origin, possibly the result of altered pressure in the labyrinth. Many deaf mutes fail to appreciate the position of their bodies, and may also be unable to co-ordinate their movements properly; the failure to maintain their balance is most marked in the dark, though it may be quite evident without the exclusion of light.

It is uncertain how many of the cases in which giddiness is an accompaniment of gastric disturbance are to be ascribed to the visceral disorder as the sole cause, in that a large number of cases formerly so regarded are now attributed to labyrinthine disturbances, nevertheless gastric and intestinal disturbance may excite an attack of vertigo in one so predisposed. The frequency with which vertigo is met with in association with gastro-intestinal affections, without any other evidence of labyrinthine disease, suggests the possibility that disturbance of equilibration may arise from this cause independently of any affection of the labyrinth. Vertigo may further be a symptom in many other affections, as for instance in neurasthenia, hysteria, anæmia, and so forth, while in some cases no ascertained or probable cause is forthcoming. A curious class of cases in point is that in which sudden attacks of vertigo occur from time to time, it may be attended with vomiting and various vaso-motor phenomena, while in the intervals there is a complete freedom from any like manifestations.

Equinia. *See* Glanders, Farcy.

Equinus. *See* Deformities (*Club Foot, Talipes Equinus*).

Erasion.—Scraping away the diseased structures in a joint or removing them with knife or scissors, the articulation having first been opened into (arthrectomy). *See* Ankle-Joint, Region of, Operations (*Erasion of the Ankle-Joint*); Knee-Joint, Diseases of (*Arthrectomy of the Knee, Erasion*).

Erb's Paralysis.—Paralysis due to injury of the brachial plexus (*e.g.* during the birth of an infant) in its fifth or fifth and sixth roots. *See* Brachial Plexus, Surgical Affections.

Erb's Paraplegia.—Syphilitic spinal paralysis, perhaps due to a form of transverse myelitis. *See* Paralysis (*Spastic Paraplegia, Syphilitic Paraplegia of Erb*).

Erb's Sign. *See* Tetany (*Motor Signs, Electrical Reactions*).

Erb-Goldflam's Syndrome. *See* Myasthenia Gravis.

Erectile Tissue. *See* Bone, Diseases of (*Erectile Tumours of Bone*); Nose (*Affections of Septum Nasi, Hypertrophy of Erectile Tissue*).

Eremophobia.—Morbid fear of solitude (Gr. ἐρημος, solitude, and φόβος, fear).

Erepsin.—An enzyme which splits up peptone and other proteids into their non-proteid crystalline constituents, such as the di-amido acids and non-amido acids. Its use in cases of cancer has been suggested. *See* Physiology, Food and Digestion (*Secretion of the Intestinal Wall*).

Erethism.—Morbid irritability or excitement; also, delirium tremens.

Ergograph.—A work-measurer, *e.g.* a wheel turned against measured resistance. *See* Physiology, Tissues (*Muscle Work, Relation of Heat Production to Work Production*).

Ergophobia.—Dr. W. D. Spanton defines *ergophobia* as follows (*Brit. Med. Journ.* i. for 1905, p. 300): "The working man . . . has discovered that it often pays better to idle and loaf about than to work, and the consequence is that a new disease has been engendered, which I have termed 'ergophobia.'" *See also* Malingering.

Ergot. *See also* Aneurysm (*Treatment, Injection of Ergotin*); Colour Vision (*Acquired Colour-Blindness*); Drug Eruptions (*Ergot*); Gangrene (*Varieties*); Insanity, Etiology of (*Toxic Causes, Exotoxic*); Insanity, Nature and Symptoms (*Etiological Varieties, Toxæmic*); Labour, Precipitate and Prolonged (*Effect of Ergot in Obstructed Labour*); Labour, Post-Partum Hæmorrhage (*Treatment*); Purpura (*Symptomatic, Toxic*); Raynaud's Disease (*Diagnosis, Ergotism*); Spasm (*Varieties, Toxins*); Tetany (*Causation, Chronic Ergotism*); Toxicology (*Abortifacients, Acute and Chronic Ergot-Poisoning*).—The sclerotium of the fungus *Claviceps purpurea*, found in the form of black horny-looking masses about one inch long on the common rye (secale cereale). It decomposes even when whole, and should therefore be used as fresh as possible. *Dose*—20-60 grs. of recently prepared powder infused in boiling water. The most important constituents of ergot are: 1. *Sphacelinic acid*, causing a tonic contraction of the uterus, and constriction of blood-vessels; 2. *Cornutine*, said to produce rhythmic contractions of the uterus; 3. *Ergotinic acid*, a glucoside with little action; 4. *Ergotinin*, an alkaloid.

Various other principles have been described, but it is not certain that these are not merely derivatives of the above. *Preparations*—1. Extractum Ergotæ Liquidum. *Dose*—10-30 m. 2. Extractum Ergotæ, commonly known as *Ergotin*. *Dose*—2-8 grs. 3. Injectio Ergotæ Hypodermica, containing 33 per cent of the extract. *Dose*—3-10 m. 4. Infusum Ergotæ. *Dose*—$\frac{1}{2}$-2ʒ. 5. Tinctura Ergotæ Ammoniata. *Dose*—$\frac{1}{2}$-1ʒ. Owing doubtless to the difficulty of obtaining the official preparations in a fresh and active state numerous other forms are now in use. *Cornutin* and *ergotinin* have both been employed, the latter usually as its soluble *citrate* in doses of $\frac{1}{150}$-$\frac{1}{30}$ gr. "*Ergot aseptic*" is a proprietary preparation of the liquid extract, concentrated and sterilised, and put up in 1 cc. glass bulbs.

The most important use of ergot of rye is to stimulate the contractions of the uterus after childbirth with a view to the prevention or arrest of post-partum hæmorrhage. Some physicians give it as a routine practice, even in the absence of any indications, but this is probably unnecessary. In some cases, however, although there are no immediate signs of hæmorrhage, it is wise to administer it, for example, after a long tedious labour, when the uterine muscle may be expected to be lax and atonic. To ensure prompt action it is usually best to employ a preparation of ergotin injected deeply into the gluteus maximus. As a general rule, ergot is to be absolutely prohibited during the second stage, as it brings about a tonic persistent contraction of the uterus, endangering the life of the child, and even in some cases acting as a contributory cause to rupture of the uterus. Occasionally one may venture on its use at this stage, but only when the cervix is fully dilated, and when one is perfectly certain that no abnormality or obstruction to delivery is present. It should practically never be employed during the third stage, the indications in almost every instance being for its administration after, and not before the removal or expulsion of the afterbirth. During the puerperium it is of value in cases of imperfect involution, or where symptoms exist pointing to the retention of blood-clots or pieces of membrane and placenta in the uterus. In accidental hæmorrhage it is good practice, after the membranes have been ruptured, to give ergot, as our object in such cases is to control the bleeding by all means that increase the contraction of the uterus. Ergot is very useful in some forms of menorrhagia. As a general hæmostatic ergot has now to a large extent fallen into disrepute. It is argued, and with good reason, that the general rise of blood-pressure produced will more than counteract any local action that may occur. When one main object in treatment is to keep the blood-pressure low, as in cerebral hæmorrhage, it is certainly to be avoided, but many authorities still strongly advocate its use in large, frequently repeated doses in hæmatemesis and hæmoptysis. When the bleeding is from an erosion in a vessel of any size ergot is obviously useless, but it is for the oozing of blood from a larger area that the drug is specially recommended, and should be given a fair trial. Headache, migraine, infantile paralysis, locomotor ataxia, aneurysm, and many other diseases have been treated by ergot at one time or another, but without any appreciable benefit. The whole question of blood-pressure and the reciprocal relations of the different parts of the vascular system is at present being investigated afresh, and many old founded beliefs are receiving a rude shock; and it is more than possible that with a better understanding of the physics of the circulation, and improved means of clinical observation, a larger sphere of usefulness in general therapeutics may be opened up for ergot of rye.

Ergotin. *See* Ergot.

Ergotinin. *See* Ergot.

Ergotism. *See* Ergot; Toxicology (*Acute and chronic ergot poisoning*).

Erlenmeyer's Method.—Rapid demorphinisation in the treatment of morphinomania. *See* Morphinomania (*Treatment*).

"Ernutin."—One of Burroughs, Wellcome, and Co.'s preparations; "a new product representing the active therapeutic principle of ergot."

Erosion.—A superficial loss of tissue, *e.g.* of the skin or of a mucous membrane. *See* Stomach and Duodenum, Diseases of (*Pathology, Ulceration*).

Erotomania.—Insanity in which there is loss of control over the sexual appetite; Nymphomania or Satyriasis. *See* Insanity, Nature and Symptoms (*Insane Defects of Inhibition, Nymphomania*); Tabes Dorsalis (*Symptomatology, Genital Organs*).

Eructations.—The bringing up of gas from the stomach. *See* Indigestion (*Special Forms, Flatulent Dyspepsia*); Stomach and Duodenum, Diseases of (*General Symptomatology, Eructations*).

Eruption.—The breaking out of a cutaneous rash or the emergence of a structure (*e.g.* a tooth). *See* Dengue (*Clinical Features, Third Stage*); Dermatitis Traumatica et Venenata (*Feigned Eruptions*); Hysteria (*Symptoms, Trophic Troubles*); Measles (*Symptoms, Eruption*); Syphilis (*Cutaneous Manifestations*); Teeth (*Eruption*); etc.

Eruptive Fevers.—The exanthemata, such as measles, small-pox, scarlatina, etc. (*q.v.*).

Erysipelas.

See also Alopecia (*Etiology*); Bone, Diseases of (*Acute Suppurative Osteomyelitis, Diagnosis*); Brain, Inflammations (*Acute Encephalitis, Etiology*); Diphtheria (*Diagnosis from erysipelas faucium*); Glanders (*Symptoms in Man, Diagnosis*); Herpes (*Diagnosis*); Immunity (*Swine Erysipelas*); Joints, Diseases of (*Pyogenic*); Leucocytosis (*Causal Varieties*); Meninges of the Cerebrum (*Meningitis from Erysipelas*); Nephritis (*Etiology*); Nephritis (*Chronic, Secondary Inflammations*); Nerves, Multiple Peripheral Neuritis (*General Etiology*); Nose, Accessory Sinuses, Inflammation (*Etiology*); Pericardium, Diseases of (*Pericarditis as Complication of Erysipelas*); Pneumonia, Clinical (*Etiology, Erysipelas*); Pregnancy, Affections and Complications (*Fevers, Erysipelas*); Purpura (*Infections*); Therapeutics, Serum Therapy (*Antistreptococcus Serum*); Tumours, Inoperable, Treatment of ("*Coley's Fluid*," *q.v.*); Urine, Bacteria in (*In Infective Diseases*).

Erysipelas is an acute, specific, spreading inflammation of skin or mucous membrane. The disease shows a marked disposition to remain localised to the skin and immediately subjacent tissue, and there is no natural tendency to pus-formation, ulceration, or gangrene. The inflammatory process usually ends in spontaneous resolution without leaving any permanent structural change in the affected tissues.

Etiology and Morbid Anatomy.—Fehleisen (1884) was the first to show that erysipelas is always associated with the growth in the minute lymph channels of a streptococcus, which he believed to be specific as regards this disease, and called the *Streptococcus erysipelatis*. For some years this view was current, but the majority of bacteriologists now hold that the disease is caused by the ordinary *Streptococcus pyogenes* "of a certain degree of virulence," although the conditions which so modify the organism as to produce this particular degree of virulence are as yet undetermined.

The organisms always gain entrance through a wound or abrasion of the skin or mucous membrane, but the lesion may be so trivial as to be entirely overlooked. A slight scratch, an acne pustule, a crack on the nasal or buccal mucous membrane, or the open cavity of a carious tooth, may be the seat of infection, and so numerous are the possible avenues that the existence of an *idiopathic* form of erysipelas is more than doubtful.

The streptococci are found in the lymph spaces of the skin, and may spread into the more superficial layers of the subcutaneous tissue. They also occur in the serous blebs which sometimes form over the inflamed area, but they never invade the blood-vessels. They are most active in the zone just beyond the swollen margin of the inflamed area, but rapidly disappear as the inflammation subsides.

On microscopic examination of a portion of erysipelatous skin four distinct zones are to be recognised:—(1) Towards the spreading margin just beyond the reddened area is a zone in which all the lymphatics are found full of rapidly multiplying streptococci. (2) Behind this the tissues show all the evidences of acute inflammation with dilated vessels, exudation of serum and migrated leucocytes, many of which contain streptococci in their anterior. (3) Still farther back the inflammatory reaction is passing off, and the leucocytes are being destroyed by the fibroblasts or macrophages, no free cocci being visible. And (4) behind this the skin has regained its normal appearances.

The organisms may be stained by Gram's method; or cultivated on gelatine or agar (*see* "Micro-organisms").

When suppuration ensues on an attack of erysipelas it is in all probability the result of a mixed infection with one or other of the pyogenic cocci.

The systemic symptoms in erysipelas are due entirely to absorption of toxines, as the presence of the bacteria in the blood has not been demonstrated. The immunity produced by one attack is very transient.

In fatal cases the *post-mortem* findings are those common to all forms of acute blood poisoning; a dark, tar-like state of the blood; a swollen and softened condition of the spleen; cloudy swelling of the heart-muscle, the liver, and the kidneys; multiple ecchymoses in the pleura, pericardium, and various mucous membranes; and dropsical effusions into the lung tissue.

Erysipelas has been inoculated as a *therapeutic measure* in various diseases, such as cancer, sarcoma, lupus, syphilitic lesions, etc., but without such a degree of benefit as to justify the risk.

Clinical Features.—The incubation of the disease was found to vary from 15 to 61 hours in a series of cases inoculated by Fehleisen for therapeutic purposes.

The first clinical sign is a feeling of malaise, with headache and pains in the back and limbs. The appetite is lost and the patient usually becomes sick and vomits. A feeling of chilliness, seldom amounting to a distinct rigor, ensues, and concurrently the temperature rises to 103°, 104°, or 105° F., and remains high with but slight remissions during the course of the disease. The pulse at first is full and bounding, and

seldom exceeds 100. The tongue is foul, the breath heavy, and the bowels usually constipated, although in some cases there is diarrhœa with offensive, dark-coloured stools.

Locally spots of redness appear near the seat of infection, and rapidly coalesce into a diffuse red patch, from which thin red streaks may pass towards the nearest lymphatic glands, which are found to be enlarged and tender.

The reddened area, which varies in hue from a bright scarlet to a dull brick red, gradually increases in size, spreading now in one direction and then in another, with varying rapidity. The spread does not follow the direction of the lymph stream, but is supposed to be regulated to some extent by the linear furrows of the skin; and certainly it seems often to be arrested at points where the skin forms deep attachments to underlying fasciæ, *e.g.* at Poupart's ligament, in the palms and soles, at the chin and nape of the neck.

The margins of the patch are slightly raised above the surrounding skin, as may be recognised by lightly passing the fingers over the skin from the healthy on to the inflamed area. The whole surface is œdematous, especially in parts where the subjacent cellular tissue is lax and open, as in the eyelids, scrotum, labia, or on the chest wall. The skin is smooth, tense, and glossy, and blebs containing clear yellow serum frequently form over areas of intense inflammation. The deep lymphatic trunks may often be felt as firm cords.

The patient complains of a burning sensation in the skin, and the inflamed area is tender to the touch. It is found on careful examination that the most tender area lies in a zone about half an inch beyond the raised red margin, a point of some clinical importance, as indicating the extent and direction of the spread, which is often found to take place irregularly.

The temperature of the affected part is markedly raised.

The usual gastro-intestinal derangements, associated with infective and pyrexial conditions, are present; and the urine is scanty, high-coloured, loaded with urates, and often albuminous. There is often pain over the kidney, and the spleen may be found to be enlarged.

Nocturnal delirium, with restlessness and delusions, is common, especially when the disease attacks the face or scalp.

The duration of the disease varies from two to three days to as many weeks; and relapses are by no means infrequent, each recrudescence being accompanied by a fresh rise of temperature.

Death may take place from toxæmia, from the process spreading to important organs, such as the brain or its membranes, or from exhaustion when the disease is prolonged or when relapses occur.

Clinical Types of Erysipelas.—Certain distinctive clinical features are to be recognised in connection with erysipelas according to—(1) the severity of the attack; (2) the part of the body and the structure of the tissues implicated; and (3) various aberrant characters it may assume.

1. (*a*) In its mildest or *erythematous* form the disease produces a diffuse superficial reddening of the skin, with but slight swelling. There are no bullæ formed, and desquamation does not follow.

(*b*) When the inflammation is more intense the *bullous* variety of the disease is produced, in which blisters containing a clear yellow serum form under the cuticle. The contents of these bullæ may readily become infected with pyogenic bacteria from the skin, and form the starting-point of a diffuse septic cellulitis. When the contents of the bullæ are hæmorrhagic it indicates a grave prognosis.

(*c*) In rare instances the inflammatory reaction is so intense that patches of the skin become gangrenous (*gangrenous erysipelas*).

2. (*a*) The *face* is the commonest situation on the body for erysipelas (*facial erysipelas*). It usually begins about the alæ of the nose, and spreading over the face, assumes a "butterfly" outline. The scalp is as a rule implicated as far back as the nape of the neck, but the disease usually stops at the chin, and almost never extends on to the front of the neck. There is great œdematous swelling of the eyelids, so that the eyes are often completely closed; and bullæ are liable to form on the cheeks and brow. Epistaxis is a not infrequent symptom. Meningitis and sinus thrombosis are dangers to be reckoned with, and may be suspected when vomiting with constant headache, irritability, and nocturnal delirium occur. Stupor or convulsions ensue in fatal cases. Transient mental aberrations, peripheral neuritis, and ataxic symptoms frequently follow on recovery from these complications. The process may spread to the eye or ear, and produce more or less serious disturbance of function in those organs.

(*b*) In infants erysipelas sometimes attacks the region of the umbilicus before the stump of the cord has separated, and may spread widely over the trunk (*E. neonatorum*).

(*c*) When the disease affects the *scrotum* a diffuse boggy swelling of the whole perineum results, and the local appearances of extravasation of urine are closely simulated. Pyogenic infection is common, leading to cellulitis and sloughing.

(*d*) Any *mucous membrane*, but especially those adjacent to skin surfaces,—such as the buccal, nasal, pharyngeal, rectal, or vaginal,—are liable to erysipelatous inflammations, which may originate on either the cutaneous or the mucous surface.

The most important variety of this class is *erysipelas of the fauces*, because of the danger of

the swelling passing to the air-passages and inducing œdema glottidis. Sloughs of the mucous membrane leaving putrid ulcers, and causing enlargement of adjacent lymphatic glands, are common.

(*e*) *Erysipelas in Wounds.*—The healing of an aseptic wound is not necessarily interrupted by a mild attack of true erysipelas. In most cases, however, the inflammatory effusions separate the edges, and pus subsequently forms in the wound.

A granulating wound becomes dry and ceases for the time being to secrete pus, or it may become covered over with a greyish false membrane.

3. (*a*) By *metastatic erysipelas* is meant a variety in which the disease appears at seats far removed from the primary point of infection. (*b*) *Erysipelas migrans* is a form of the disease, which, though mild in degree, tends to wander over very wide areas of the body. (*c*) The term *habitual erysipelas* is applied to cases where the patient is liable to have repeated attacks, usually occurring about the same time of year, and in the same situation on the body.

Complications of Erysipelas.—The most important local complication of erysipelas is the occurrence of a *diffuse suppurative cellulitis*, which may spread into the connective tissue planes and lead to gangrene of various soft tissues, especially fasciæ and tendons. This condition is due to the entrance of pyogenic bacteria, and supervenes most frequently on erysipelas originating in a surgical wound. Its onset completely changes the clinical picture, and the signs and symptoms of the cellulitis completely overshadow those of the erysipelas. (*See* "Suppuration.")

It is not uncommon to find many small *localised superficial abscesses* develop during the convalescent stage of erysipelas after all the acute inflammatory symptoms have subsided. These are in all probability due to the action of pyogenic organisms which have been present in the deeper layers of the skin, and have found in the devitalised tissues suitable conditions for their development. The rapidity with which such abscesses heal after being incised is remarkable.

In the female, erysipelas in the region of the pudenda may spread to the uterus and its adnexa. This is especially liable to occur during the puerperal state, and to give rise to one of the most serious forms of *puerperal sepsis*.

In a few rare cases a peculiarly persistent form of *œdema*, almost like an elephantiasis, of the affected part may result.

The occurrence of *intracranial complications* and of implications of the *organs of special sense* have already been referred to.

Differential Diagnosis.—The diagnosis of true and uncomplicated erysipelas is as a rule easy. The conditions with which it is liable to be confused are—

(1) *Simple Erythema, Acute Eczema*, or *Acute Dermatitis.*—These inflammatory skin affections are mainly local in their manifestations, and only in severe cases give rise to a marked degree of constitutional disturbance.

(2) *Erythema nodosum* is usually in patches, bilateral, and associated with rheumatism in young women.

(3) *Severe herpes* may at first simulate erysipelas.

(4) *Lymphangitis* occurs in streaks along the lines of the main lymphatic trunks, which may be felt as firm cords.

(5) *Thrombo-phlebitis* also manifests red lines with hard cords along the course of the affected tissue, and is often associated with a septic ulcer.

(6) *Acute suppurative cellulitis*, and (7) *acute spreading gangrene*, or even (8) *acute osteomyelitis*, may at first sight be mistaken for erysipelas, but careful clinical investigation should prevent serious errors in diagnosis.

The Prognosis of uncomplicated erysipelas is on the whole hopeful, and is based upon the usual considerations as to the site, severity, extent, and duration of the disease, and the age and general condition of the patient. The prognostic significance of complications is obvious after what has already been said.

Treatment.—That erysipelas has become so much less frequent since the introduction of antiseptics is sufficient ground for demanding the systematic use of these agents with a view to preventing its occurrence.

The *indications* for treatment in an active case of the disease are—(1) to prevent its further spread; (2) to guard against mixed infection; (3) to allay local symptoms; (4) to counteract general constitutional disturbance; (5) to treat special symptoms.

1. The spread of the disease being due to the growth and multiplication of the causal organisms, it is evident that it can only be arrested by attacking and destroying these, and many attempts have been made to this end. The local application of strong antiseptic lotions, especially such as have the power of penetrating the skin, like carbolic acid (1 in 20) or corrosive sublimate (1 in 1000); the inunction of mercurial ointment; the tight application of bands of adhesive plaster with a view to occluding the lymphatics, have all been tried with more or less success.

Believing that by the phagocytic action of the leucocytes the spread of the organism is arrested in the natural cure of the disease, the writer some years ago employed a method of treatment with considerable success, the object of which is to produce a zone of increased leucocytosis *in front of* the advancing streptococci. The method consists in painting round the

erysipelatous area a ring of *linimentum iodi*, about half an inch wide. This ring is applied about one inch from the margin of the affected skin, the limit of the disease being reckoned by the tenderness on slight pressure which is usually found to be well in front of the red area. Several coats of the iodine are painted on one after the other dries. When any doubt exists as to whether the limit of the disease has been reached a second ring is applied about an inch beyond the first. The painting should be repeated for two or three days, even although it has never been crossed by the erysipelas.

Scarification of the skin in front of the spreading margin doubtless acts in the same way, but is painful and necessitates a general anæsthetic. The injection of carbolic acid under the skin has not been satisfactory.

2. Systematic antiseptic precautions in dealing with wounds, blebs, etc., will tend to prevent infection with pyogenic bacteria.

3. The most soothing *local applications* are ichthyol ointment (1 in 6) applied on lint and renewed daily; thiol in 20 to 40 per cent acqueous solution painted over the whole of the reddened area and for some inches beyond it several times a day; or simple lead and opium fomentations. After the inflammation has ceased, and while the desquamation of the epidermis is going on, the part should be smeared daily with a mildly antiseptic and soothing ointment, such as ichthyol, eucalyptus, or weak boracic acid to allay the itching which is often troublesome, and to prevent the dissemination of germ-laden scales.

4. *General treatment* consists in administering a sharp mercurial or saline purge at the outset, and attending to all the other emunctories. Quinine in two-grain doses every four hours is useful; but the value of perchloride of iron is doubtful. It frequently deranges digestion and often produces constipation. A full fluid dietary is necessary. The *antistreptococcic serum* of Marmorek promises to be useful in counteracting the toxines in this as in other diseases due to the streptococcus. In this country the experience of its action has as yet been small and not altogether encouraging as to results, but those who have used it most abroad speak favourably of it. An initial dose of 20 c.c. of the serum should be injected under the skin of the abdomen, and further doses of 10 c.c. may be given every 12 hours till the temperature comes down.

5. Special symptoms, such as delirium, sleeplessness, nephritis, or other complications, will be treated on general principles. Suppurative complications must be met by early and free incisions, with drainage and the use of antiseptic dressings frequently renewed.

Erythema.

See also Bromism (*Cutaneous System*); Chorea (*Complications, Erythema Nodosum*); Dermatitis Herpetiformis (*Types, Diagnosis*); Diabetes Mellitus (*Symptoms, Complications, The Skin*); Dressings (*Salicylic Acid, Iodoform, etc.*); Drug Eruptions (*Erythematous*); Gout (*Irregular, Cutaneous System*); Herpes (*Herpes Zoster, Initial Lesions*); Influenza (*Effects on Different Systems, The Skin*); Leprosy (*Clinical Features, Lepra Tuberosa*); Lupus Erythematosus; Malingering (*Varieties, Cutaneous*); Meningitis, Tuberculous (*Etiology, Erythema Nodosum*); Meningitis, Epidemic Cerebro-Spinal (*Symptoms, Skin Lesions*); Muscles, Diseases of (*Polymyositis Hæmorrhagica*); Nerves, Multiple Peripheral Neuritis (*Symptomatology*); Pellagra (*Symptoms, Skin Eruptions*); Pemphigus (*Pemphigus Vegetans, Diagnosis*); Pharynx, Chronic Infective Diseases (*Erythema of Fauces*); Rheumatism, Acute (*Symptoms and Course, Erythema Nodosum*); Rheumatism in Children (*Symptoms, Skin Affections*); Rheumatism, Chronic (*Clinical Features, Erythema*); Sclerodermia (*Diffuse*); Scrotum, Diseases of (*Elephantiasis Scroti*); Skin, Tuberculosis of (*Erythema Induratum*); Skin, Bacteriology (*Erythema*); Small-Pox (*Symptoms, Prodromal Rashes*); Stomach and Duodenum, Diseases (*General Symptomatology, Remote Symptoms*); Therapeutics, Serum Therapy (*Antidiphtheritic Serum, Results*); Typhoid Fever (*Complications and Sequelæ, Cutaneous System*).

The term Erythema should properly indicate merely a redness or hyperæmia of the skin. Since, however, the name has been given to a variety of eruptions, most of which also show either serous exudation or cellular infiltration of the skin, it is now used in a manner which is completely arbitrary and justified only by custom. All attempts at a natural classification must be at present unsatisfactory, on the one hand because there are still such formidable gaps in our knowledge of the relationship between allied forms, and on the other because a number of completely independent diseases are also collected under this name. The method adopted here, therefore, will be to give, first, a short description of those forms which are the result of local irritation and are of limited extent; secondly, to discuss the generalised erythemata; and, thirdly, to describe those diseases,

mostly of unknown pathology, to which the name erythema has been given, and which have no known relationship to the preceding forms.

I. Erythema due to Local Irritation and of Limited Extent.—(*a*) From heat (*E. caloricum*); (*b*) from light (*E. solare*).—Neither of these two forms requires any further discussion here, as both are merely the first stages of acute dermatitis. (*c*) From friction (*E. paratrimma*).—This term, though if it were used correctly it would be limited to erythema from friction, is more generally applied to the hyperæmia occurring in parts which have been subject to pressure when that pressure is removed, *e.g.* the sacrum and trochanters of a bedridden patient. If the affected skin is not carefully looked after in such cases by relieving the pressure with suitable pads, water-bed, etc., the process will rapidly pass on into local gangrene and bedsore. (*d*) From friction combined with chemical irritation (*E. intertrigo*).—In this form the skin is irritated partly by the rubbing of one surface against another, partly by the decomposition of the secretions which are locked up between warm cutaneous folds and prevented from evaporating. If left unattended to the parts soon become acutely inflamed, and a condition indistinguishable from eczema is set up (*Eczema intertrigo*). All that is necessary in the early stage is to carefully cleanse the parts and dry them, afterwards keeping them separated by means of suitable powders and lint, or by interposing one of Unna's powder bags between the contiguous surfaces. The disease occurs most commonly between the thighs and nates of children, or beneath the breasts and abdomen of fat adults. Closely allied to this form is the *Erythema gluteale* of infants, which is produced not so much by friction of opposing surfaces of skin as by the constant maceration of the skin by the decomposing urine on the diapers. This form has some importance, first, because of its liability to go on to troublesome inflammation and even ulceration if neglected; secondly, because of the extreme difficulty which is often found in distinguishing it from congenital syphilis. The points to be attended to are the following:—Diaper rash seldom extends below the knees, while syphilis is very likely to be found on the soles of the feet; diaper rash begins as minute papules with little or no induration, while syphilis usually shows itself as rather larger papules with very definite induration. Lastly, if no other indications of syphilis are present the child should be treated with some non-mercurial local application, such as Lassar's zinc paste,[1] and the further evolution of the rash watched for a day or two. (*e*) From the effects of locally applied drugs and other irritants (*E. venenatum*).—*See* "Dermatitis venenata." (*f*) From cold.—This may be localised or generalised, and is usually seen as a passive hyperæmia due to the contraction of the cutaneous arterioles and consequent stagnation of the blood in the capillaries from diminution of the *vis a tergo*. After the arterial spasm has passed off, a strong, active hyperæmia usually sets in, causing the original cyanotic mottling (*Livedo annulare*) to disappear and to be replaced by a general hot redness of the whole body or the part affected. The same train of symptoms is said to be occasionally seen from exposure to heat. (*g*) From circulatory obstruction (*E. leve*).—This form is often seen in patients suffering from cardiac weakness and œdema of the legs. Its significance is ominous, since, if the legs are not quickly relieved by draining the fluid away under the strictest antiseptic precautions, cellulitis and gangrene are almost certain to occur.

[1] ℞ Acid. salicyl. gr. x., Pulv. amyli. Zinci oxidi āā ʒij., Vaseline ℥ss.

II. Generalised Erythemata.—(A) *Roseolous and scarlatiniform erythemata.*—Some authors have attempted to separate these two forms, placing the former under the heading of congestive, and the latter under exudative erythemata. There seems, however, to be no reason for this either in their clinical appearance or in their pathology, so far as it is known. Roseola is the name applied when the rash appears as rather large macules, thus simulating measles. It is commoner in children than in adults, probably owing to the greater excitability of the central nervous system. There is generally some *malaise* preceding the eruption, and often some slight elevation of the temperature. The rash may come out on any part of the body or limbs, having no site of predilection such as is usually found in measles. In some cases the rash never completely generalises, remaining more or less limited to the site where it first appears. The macules are usually from the size of a lentil to that of the thumb-nail, and are arranged either irregularly or in crescentic form. The diagnosis has to be made from measles and from rötheln, and is by no means always easy. From the former the points of distinction are:—The constitutional disturbance is not usually so severe in roseola as in measles, and the other symptoms of measles, coryza and conjunctivitis, are absent. Further, if the case be seen early, or the history be very clear, it may be possible to find out the site first attacked by the eruption; or again, should the eruption never become generalised this would be of assistance. From rötheln the diagnosis is apt to present still more difficulty, and must be often impossible; but the enlargement of the occipital and mastoid glands, especially if any history of exposure to infection were forthcoming, would help in the diagnosis.

Erythema scarlatiniforme occurs more frequently in adults. The prodromal symptoms, when present, are similar to those of the

roseolous form, but there is a tendency for them to be rather more severe in character, and this is especially true of the accompanying sore throat. The rash differs from that of roseola only in its elementary lesions, the capricious distribution being found in the scarlatiniform just as was described in the roseolous. At the beginning of the eruptive stage the rash is seen to consist of well-defined punctiform macules, quite indistinguishable from those of scarlatina, and these soon run together to form large sheets of continuous redness. The differential diagnosis from mild cases of scarlatina is often extremely difficult, but the following points may prove of service. The prodromal stage of scarlatina is usually much more severe and of more definite duration than that of E. scarlatiniforme. The constitutional symptoms are more severe throughout in the specific fever, and the throat is much more swollen, tonsillitis being very rare in the scarlatiniform erythema. The duration of the eruption may be the same (two to six days) in the two diseases, but in E. scarlatiniforme the rash generally turns to a peculiar brownish yellow colour as it fades, and Besnier has called attention to the fact that desquamation is already beginning while the rash is at its height. Lastly, the desquamation is generally only branny in character in the erythema, though in severe cases the horny layer may be shed in larger casts.

Mention must here be made of a disease described by Féréol in 1876 under the name of "Pseudo-exanthème scarlatiniforme recidivant," but now more generally called "Erythema scarlatiniforme desquamativum." The true position of this disease is still a matter of some doubt, some authorities placing it with Dermatitis exfoliativa, while others describe it with the erythemata. The disease commences with general malaise, and sometimes with chills, anginal sore throat, and albuminuria. The rash comes out exactly similar to that of E. scarlatiniforme, but instead of dying away in a few days it often persists for a month, or even longer, the temperature usually falling while the eruption is still at its height. Desquamation is generally very marked, and occurs in coarse flakes. The disease has a strong tendency to recur in the same individual, chiefly in the spring and autumn.

The etiology, pathology, and treatment of these forms of generalised erythema are so closely allied to, if not identical with those of Hebra's Erythema multiforme that it will save repetition if they are all discussed together.

(B) *Erythema exsudativum multiforme.*—This group, probably the most important of all the erythemata, we owe to the genius of Ferdinand Hebra, who collected a series of eruptions known by different names, and pointed out that they were all stages of the same disease. Although his conception was perhaps too circumscribed, and has consequently been slightly amended, it remains to this day the basis upon which all descriptions of the disease are founded. For convenience of arrangement the different forms of eruption will first be described individually, then the symptoms and complication in other organs, and pathology, will be discussed for all forms together.

Types of Eruption.—(*a*) *Erythema papulatum.*—This may be considered as the simplest form of eruption occurring in E. multiforme. It is characterised by the sudden outbreak of papules varying in size from that of a large millet seed to that of a split pea. The papules are formed by exudation into the corium and upper part of the hypoderm, and are elevated above the general cutaneous surface in hemispherical form. They are moderately hard to the touch, and the epidermis over them is tense and shining. Their colour varies from a florid red on their first appearance to a yellowish red in the centre of old papules, or sometimes a bluish, cyanotic appearance. The rash fades completely on pressure at first, but as there is often some diapedesis of red blood corpuscles later on there is a slight yellowish stain left after the hyperæmia is pressed out. After the disappearance of the eruption there is often a slight desquamation of the horny layer, but this is not a marked feature. The distribution is most commonly on the upper and lower extremities, the backs of the hands and feet, though these are by no means invariably attacked, as has been stated, and the face and neck, other parts, such as the trunk, being less often attacked. The eruption does not usually become confluent, and is usually scattered without any definite arrangement beyond following the lines of cleavage, but sometimes a crescentic arrangement may be observed.

(*b*) *E. tuberculatum.*—This form may arise either primarily or by the centrifugal extension of the papules of the previous form. The lesions are larger and are situated rather deeper in the corium, thus possessing a more spherical shape; they are also usually rather harder than those of E. papulatum, owing to the greater amount of exudation into the surrounding tissues. The site, arrangement, and other characteristics are similar to those already described under E. papulatum.

(*c*) *E. annulatum.*—This is usually a secondary development from E. papulatum. The papules spread centrifugally while involution occurs in the centre, so that a red, raised ring is formed with a dusky purplish centre. Such rings in the course of their extension may show peculiar changes of colour. In the centre, as already stated, the colour is of a purple, cyanotic hue, due no doubt to the passive hyperæmia left after the absorption of the previous exudation. The raised, active part is usually of a florid red

owing to the arterial hyperæmia still present, and surrounding this there is often a yellowish, somewhat translucent zone of œdematous skin in which the blood-vessels are compressed by the exudation. This arrangement has given rise to the name *E. iris*, a term which is also applied to another form, described below, and better known as *Herpes iris.* With the progressive enlargement of the rings it is obvious that two or more rings may intersect one another, and, in obedience to the law of all centrifugally extending skin diseases, the parts of the rings within the points of contact disappear. In this way peculiar curved figures are formed, to which the name *E. gyratum* has been given. In some cases this actively advancing edge may continue spreading over large areas, so that only a marginal redness is seen enclosing discoloured skin. To this condition the term *E. marginatum* has been given, but this seems to be an entirely unnecessary refinement of nomenclature.

(*d*) *Herpes iris* or *E. iris.*—This is yet another modification of the primitive papule. The essential change in this variety is the formation of a vesicle or bulla by the raising of the whole or part of the epidermis by the exudation. Two forms of the disease are generally described, both of them uncommon, but one much rarer than the other. The commoner form begins as a small papule in the centre of which a vesicle rapidly forms. In the same manner as described under E. annulatum the papule enlarges centrifugally, and the vesicle formation follows this enlargement. There is thus formed a small disc-shaped blister situated on a red base. As the lesion still further increases in size there is a tendency to resolution in the centre, which gives rise either to a purple stain surrounded by a ring-shaped vesicle, or perhaps more frequently the exact centre of the lesion is occupied by dark red scab caused by the drying up of the central vesicle, in the serum of which there were contained some red blood corpuscles. Since the vesicle in E. multiforme is generally situated rather deeply in the epidermis, a curious pearly grey appearance is seen where the exuded serum is, as is commonly the case, small in amount. There is thus produced in many cases a remarkable play of colours, from which the name Iris is derived. In the centre is seen the dark red scab, outside this is a zone of purple passive hyperæmia over which the fluid has been partially absorbed or evaporated, outside this again is the pearly grey colour of the recent vesicle, surrounded by a narrow band of active hyperæmia, and lastly by a pale yellowish areola of anæmic œdematous skin. The size of the developed lesions of this form varies from that of a threepenny piece to, rarely, a five-shilling piece. The other form is not quite so clearly related to the ordinary varieties of E. multiforme. It begins with a vesicle which enlarges to form a bulla, and is then surrounded with rows of discrete vesicles, but with little erythematous redness.

(*e*) *E. nodosum.*—The identity of this disease with E. multiforme is denied by some writers, though all admit the close relationship. The tendency at present is, however, apparently becoming more and more general in favour of including it with the forms just described. The main reason for this is its common occurrence on the lower extremities in patients who have the papular or tubercular form elsewhere, and as regards the alleged objection that E. nodosum often occurs without any of the other forms, this is true of any given type of the disease. The lesions are situated more deeply in the corium than in any of the previously-described forms, extending indeed right through the hypoderm. They appear as large nodes, varying in size from that of a small nut to that of a pigeon's egg, or sometimes even larger. The nodes vary in colour, being sometimes of a darkish purple, sometimes florid pink. Their consistency is less hard and more elastic than that of the papular and tubercular forms, and they have the additional peculiarity of being painful and extremely tender to the touch. In some cases there seems to be distinct fluctuation, and cases have been described in which they have softened and burst, but this is, at all events, extremely rare. The site of predilection is the front of the shin and lower leg generally; in fact, so much is this the case that one seldom sees really typical nodes elsewhere, though they do occur along the ulnar border of the forearm and occasionally on the face. When fading the nodes go through all the changes observed in the healing of an ordinary bruise, and hence the synonym Dermatitis contusiformis.

Duration of E. multiforme.—The individual lesions do not as a rule last more than a few days, but since successive crops are apt to come out, the disease as a whole usually lasts from two to four weeks. On the other hand, cases have been published in which the disease has lasted from two months to several years (E. perstans). As regards the relative duration of the different types of lesion, the papular usually disappears most quickly, while the nodose and bullous forms last the longest.

General Symptoms and Complications in other Organs.—The constitutional symptoms of E. multiforme are variable in the extreme. Many cases run their entire course with nothing but a little burning at the sites of the eruption, on the other hand patients are occasionally so severely affected that they pass into the typhoid state, from which, however, they generally recover. If one is justified at all in describing a typical course for such a variable disease, the following would perhaps correspond to what one might term an average case. After pro-

dromal symptoms lasting a day or two before the appearance of the eruption, loss of appetite, headache, ill-defined pains in the muscles and joints, and especially a feeling of extreme lassitude, with perhaps slight fever, the rash suddenly makes its appearance, and continues to appear in crops for one or two weeks. During this period the symptoms of the prodromal stage are usually continued, and to them is added a rather rapidly supervening anæmia. After this no fresh lesions come out, and convalescence progresses steadily. As regards the occurrence of fever, there does not seem to be any very definite relationship to the severity of the eruption, since Polotebnoff in his monograph quotes two cases, in one of which fever was severe with a slight eruption, and in the other a very profuse eruption was accompanied by only slight fever. Of symptoms occurring in other organs the commonest is the occurrence of erythematous or bullous lesions on the mucous membrane and conjunctivitis. This is the case chiefly with the vesicular forms of the disease, but nodose erythema has also been observed on the lips and palate, in the latter of which situations the nodes burst and left crateriform ulcers. Gastric and intestinal symptoms, such as vomiting and diarrhœa, are not uncommon, and may be very severe, and it is of interest to note that in a fatal case of Hebra's E. gyratum was found post-mortem in the small intestine. Besides the fleeting pains in the muscles and joints so commonly seen, there may be organic changes in the joints, usually ending in complete recovery, but occasionally leading to ankylosis. Affections of other serous membranes, such as pleurisy and pericarditis, have been frequently observed, and endocarditis may also occur. Bronchitis and broncho-pneumonia have also been reported as producing a fatal result in some cases. Swelling of the liver or spleen, or both, seems very common, and Polotebnoff quotes two cases of his own in which general enlargement of the lymphatic glands occurred.

Diagnosis.—The papular and vesicular forms of erythema have to be distinguished from measles, eczema, syphilis, and Dermatitis herpetiformis. From measles the points of distinction have already been given under roseola. Acute vesicular eczema may be generally separated by the facts that the vesicles and papules are smaller and much more closely set, thus tending to run together, that they occur in large sheets, that each individual papule or vesicle is more pointed, that in most places the follicles are especially picked out, that the subjective symptoms are more restricted to severe itching, and lastly, that constitutional symptoms are slight or absent. From syphilis the diagnosis may be so difficult that with all accompanying symptoms it is still doubtful. The greater indolence of the syphilitic lesions and the shotty lymphadenitis are chiefly to be relied upon; the throat symptoms may, it is true, be of service, but where marked affection of the throat occurs in E. multiforme the distinction will be very difficult. From a first attack of Dermatitis herpetiformis the diagnosis may well be impossible. The chief points are more itching in D. herpetiformis, wider generalisation of the rash, less often a tendency to the concentric arrangement so typical of E. multiforme, and, as a rule, at first less marked constitutional symptoms.

E. nodosum must be distinguished from gummata and from Erythema induratum. From the former the points of distinction are the more rapid formation, the greater symmetry, and usually the greater number of nodes, the much more marked tenderness, and, lastly, the less defined limits to the nodes. From the latter the points are the greater tenderness, the situation on the front rather than on the back of the leg, the more acute course, and the constitutional symptoms. In some severe cases the clinical features closely simulate acute osteomyelitis (*vide* "Bone).

Pathology and Etiology.—The collection and analysis of large numbers of cases of the various forms of E. multiforme have rendered the hypothesis that it is a specific disease untenable. Besnier has pointed out that it is important to realise that there is no specificity of cause in the erythemata. It is conceivable that the determining cause of the eruption may act in one of at least three ways, namely, on the vascular centres, on the vaso-motor nerves, or upon the vessel walls. It is probable that the rash may be produced in any one of these ways, and, in view of the fact that some individuals are much more prone to the eruption than others, it is necessary to presuppose a greater delicacy of the neuro-vascular system in such persons. This is the individual disposition assumed by Besnier, who believes that it may be innate and permanent, or acquired and transitory. Such being the case it is easy to see that irritants acting locally may produce—(1) An erythema co-extensive with the area of application, direct action on the vessel walls; (2) an erythema spreading beyond the area of application, but not generalised, action on the vaso-motor nerves and secondary ganglia; (3) a generalised erythema, due either to reflex action on the vaso-motor centre or to absorption and circulation of the irritant. The eruptions of the first and second class are usually roseolus or scarlatiniform in type, and may be produced by innumerable causes. Among others mercury and iodine employed locally are common causes, but it is to be remembered that mercury especially may produce a generalised erythema either of the scarlatiniform type or of E. multiforme, when applied locally. Lewin also reported the

artificial production of E. multiforme by irritating the female urethra with a sound and savine ointment. These cases are, however, not free from all doubt, since the patient in each case had been suffering from gonorrhœa, and the irritation may have easily caused a recrudescence of the disease and an accompanying toxæmia. The causes acting by internal absorption are :—

(1) Drugs, including antitoxins and tuberculin (*see* "Drug Eruptions").

(2) Foods, generally in a state of decomposition; the commonest being shell-fish, especially mussels, game, cheese, and canned meats. Also grain poisoning (*see* "Pellagra").

(3) Toxins from organisms existing in the body. (*a*) From acute specific fevers, especially enteric, cholera, diphtheria, variola (prodromal eruption), vaccinia, gonorrhœa, sapræmia, septicæmia, and pyæmia (this is the so-called surgical and puerperal scarlatina; for further details see under the headings of these diseases). (*b*) From chronic infective diseases, especially tuberculosis and syphilis, in which latter disease E. multiforme has several times been observed, either immediately before or after the earliest syphilitic rash.

(4) From absorption of poisons, usually shut off from the circulation, *e.g.* hydatid eruption and erythemata after enemata.

(5) From poisons generated in the body from altered metabolism, albuminuria, and uræmia, diabetes, cholæmia, etc.

(6) From seasonal and atmospheric conditions, including rheumatism. This class of cause needs a few words of explanation. In the first place, although it is not difficult to draw up elaborate etiological schemes, it will be found in the majority of cases the cause cannot be found. The disease is undoubtedly very much more prevalent at certain seasons of the year, notably spring and autumn, and in damp weather, and it may therefore be considered probable that it is due to infection of unknown origin, occurring when the bodily resistance is lowered by cooling. As regards the relationship to rheumatism the question still remains open. Owing to the frequent presence of pains in the muscles and joints, together with the occasional occurrence of endo- and pericarditis, many authors have considered the disease to be merely a symptom of rheumatism. It has been, however, pointed out that such a train of symptoms is common to many acute infective diseases, and that therefore it does not afford sufficient evidence of the presence of rheumatism. Other observers have taken the opposite standpoint, and denied all relationship between E. multiforme and rheumatism. This view can, however, only be maintained by neglecting to take into consideration the undoubted fact that there is a special liability to be attacked by E. multiforme in patients who have either been sufferers from acute rheumatism themselves, or in whose family history rheumatism is noticeably frequent. This association of rheumatism and E. multiforme is perhaps strongest in the case of E. nodosum.

(7) From specific organisms. This as a cause is probably extremely rare, but its occurrence can scarcely be denied in the light of five cases reported by Professor Demme. In these cases, all of them children, varying in age from three to eleven, the symptoms were the same in all, namely, severe bullous and nodose erythema ending in gangrene, and associated with pains in the muscles, bones, and joints. Recovery took place, but one child died eleven months later of tuberculous meningitis. Bacteriological examination showed the constant presence of a bacillus, which proved pathogenic to guinea-pigs on inoculation by scarification or subcutaneous injection, producing nodes and bullæ with gangrene. Other cases have also been reported, in which cocci have been found in the lesions, blood, etc, but they are not so convincing. Apart from their general pathological interest, these cases have a special bearing in proving that in some cases, at all events, the production of symmetrical erythemata may be brought about by the local action of microbes.

The pathological anatomy of the erythemata is comparatively simple. On section it is found that the lesion is formed by a more or less hæmorrhagic exudation into the corium, the vessels being all surrounded by a dense infiltration of cells. In bullous cases there is necrosis of almost the whole depth of the epidermis. The blood of patients suffering from E. multiforme and the serum of the bullæ generally show a condition of marked eosinophilia, but as this is also found in many other skin diseases its diagnostic significance is not great. (*See* "Leucocytosis.")

Treatment.—From what has been already said of the etiology it will be seen that in many cases the treatment resolves itself into that of the disease in the course of which the erythema arises. In those cases in which no cause can be ascertained treatment is apt to leave one quite in the lurch. The acute cases run their course so rapidly that it is often impossible to judge of the effects of the drugs administered. In those cases in which there is a history of the ingestion of unsuitable foods or of acute disturbance of the digestive organs, a single dose of calomel should be given, followed by a morning saline draught, which should be repeated for two or three days. In the more chronic cases quinine in full doses (at least 15 grs. daily) is generally of the greatest use, while in some cases sodium salicylate has been found of service. In Erythema nodosum rest in bed is an important part of the treatment, while one or two authors have claimed that potassium iodide acts as a specific.

Locally bland powders and cooling lotions are

all that are required, with the addition of careful bandaging in E. nodosum.

III. Erythema pernio (*Chilblain*).—This is an inflammatory erythema occurring chiefly on the extremities, the ears, and the nose.

The first symptom is a slight burning or itching, and this is rapidly followed by the appearance of a circumscribed, dusky-red swelling. The epidermis over the swelling is tense and shiny, and is often raised in the later stages by the exudation beneath, forming a blister with more or less hæmorrhagic contents. On rupture of the blister superficial ulceration may set in, and unless this is carefully attended to, obstinate gangrene. The pathology of the affection is unknown, some authorities ranking it with true frost-bite, while others consider that it is more nearly related to E. multiforme. Certainly the clinical appearances are very suggestive of a papular or vesicular erythema, and the disease would appear to be caused by a damage of either the vessel wall or the vasomotor nerves. Though it is commonly stated that chilblain occurs in people with a "poor" circulation and a general tendency to cyanosis of the extremities, this is by no means always the case, and a little observation will speedily convince any one that many patients with a strong tendency to blueness of the extremities never suffer from chilblain, while, on the other hand, people without any obvious circulatory defect may be attacked directly the weather turns cold.

The treatment is rather prophylactic than remedial. Patients who are liable to the disease should go about warmly clad with woollen hose and gloves in cold weather. The hands and feet may occasionally be soaked in hot mustard and water, of course when no broken chilblains are present, and the stockings may be wrung out of a weak alcoholic solution of capsicum (equal parts of tr. capsici and eau-de-Cologne).

Regular friction of the extremities is invaluable, and brisk daily exercise, especially early in the day, seems to lessen the tendency to vasomotor irritability. In the early papular stage of chilblain painting the part with tincture of iodine seems to be the most useful measure, but many other stimulating remedies have been recommended, such as soap liniment, camphorated spirit, turpentine, etc.

When broken or ulcerated, protection with some mildly antiseptic ointment, such as the B.P. boric acid ointment, should be carried out. Of internal remedies, cod-liver oil, ichthyol, arsenic, have all been recommended, but they all of them often fail.

IV. Erythema induratum (*Bazin's Disease*).—This comparatively rare disease has attracted considerable attention of late years. Though originally described by Bazin, its existence was almost forgotten until further attention was called to it by Colcott Fox. The eruption occurs chiefly in young women whose occupation necessitates long standing, such as washerwomen and shop assistants. The subjects are also generally sufferers from chilblain, and in some cases have also been attacked by lupus erythematosus. Undoubtedly some of them show signs of the so-called scrofulous diathesis, a point which attracted Bazin's attention and caused him to describe the disease as "Erythème induré des scrofuleux." The disease usually attacks the lower extremities, its commonest site being the back of the leg at the junction of the lower and middle thirds, but it may also occur anywhere on the arms or legs. The distribution is almost if not quite invariably bilateral. The lesions commence in the hypoderm as rather diffuse infiltrations, which can be felt before they are seen. As they increase in size they rise to the surface and then appear as indolent nodes of a curious violaceous colour, generally flattened on the surface, hard and somewhat tender to the touch, but not usually spontaneously very painful. In this condition they may last for weeks, finally either fading away, or softening and necrosing, so as to leave punched-out ulcers, a result first described by Jonathan Hutchinson. The resulting ulcers are then extremely slow in healing, though they do not, as a rule, show a very marked tendency to spread. The morbid anatomy and experimental pathology of this disease have been most assiduously worked at lately, but the results are still conflicting, some observers finding all the structural and experimental proofs of tuberculosis, while others found no evidence of the kind. This merely goes to prove that two diseases were examined under the same name.

The diagnosis has to be made from gummatous syphilis, the so-called tuberculous gumma, and occasionally from E. nodosum.

From the first the points of distinction are the slower evolution of the lesions, the absence of other signs of past or present syphilis, the greater symmetry of the eruption, and the failure of the therapeutic test by means of potassium iodide. From tuberculous lesions of the gummatous type the disease is distinguished by its symmetry, its invariably attacking the legs, whether elsewhere as well or not, the greater number of the lesions, the fact that the tuberculous nodes generally form abscesses, while those of E. induratum generally slough, and, lastly, the age of the patient, tuberculous gummata being generally limited to children.

V. Erythema elevatum diutinum (*Crocker*).—Under this name Crocker has described a rare disease of the hands and other parts. The eruption consists of erythematous patches and nodules situated on the fingers, knees, elbows, and buttocks. The colour varies from pink to dark purple, and dilated venules are present

here and there. The nodules vary in size and may be raised as much as an eighth of an inch above the surrounding surface, while coalescence of several nodules may lead to the formation of large patches. The affected parts are hotter to the touch than the normal skin, and the surface is generally smooth, though slight scaling may be present. Induration is very marked. Crocker has separated the cases which he has collected into two classes, in one of which the disease affected only young females with a rheumatic or gouty personal or family history, the other occurring in elderly males with a gouty personal history. In the former type the lesions were erythematous at first, then becoming purple, some involuting while others were developing, and the lesions were very firm and not œdematous. In the latter the lesions began as purple nodules, persisted through life, and were softer to the touch, the elevation being largely due to œdema. The histology of the former type was simply a fibro-cellular structure more or less like cheloid. Some cases appeared to derive benefit from arsenic, but Crocker himself points out that the improvement may have been spontaneous, since some of the lesions were undergoing involution before the commencement of the treatment. The lesions on the hands seemed far more resistant than those elsewhere.

VI. Erythema serpens (Morrant Baker) (synonym, *Erysipeloid Rosenbach*).—This is a disease chiefly seen in those who handle animal food, especially game. It nearly always commences as an inflammation round a trifling injury which has been received from a few days to a fortnight before. There is an inflammatory pinkish blush surrounding the site of the previous trauma, which is itself often already healed. The erythema spreads centrifugally as a ring from this point, and later on breaks up into irregular curves and blotches. The sites are usually the finger-joints and knuckles. Although there is but little swelling and the objective symptoms are very slight, lymphangitis being practically absent, there is marked burning and shooting pain, often radiating up the arm, and the patient bends the finger gingerly. The disease lasts, perpetually extending centrifugally, for from two to six weeks, when it dies away, leaving no after-effects. The treatment recommended is salines internally and fomentations locally. Rosenbach found in his cases an organism which he first believed to be a coccus, but later classified as a cladothrix. From pure cultures of this organism he succeeded in reproducing the disease after an incubation period of forty-eight hours.

Erythema Infectiosum. *See* Fourth Disease.

Erythræa.—Various species of Erythræa (such as the Centaury) belonging to the order of the Gentianeæ have been used medicinally as tonics, as febrifuges, and in dyspepsia.

Erythrasma.—A contagious skin disease due to a fungus, the *microsporon minutissimum*. *See* Skin; Parasites (*Tinea Erythrasma*).

Erythrite. — A tetrahydric alcohol, $C_4H_6(OH)_4$, found in Roccella tinctoria, etc.; Lichen sugar.

Erythroblasts.—Rudimentary (nucleated) red blood corpuscles. *See* Blood (*Corpuscular Elements and Granules*).

Erythrochloropsy. — Blue-blindness, only red and green being distinguished. *See* Colour Vision (*Colour Blindness, Blue-Blindness*).

Erythrocytes.—The red corpuscles of the blood. *See* Blood (*Corpuscular Elements*); Physiology, Blood (*Cells of the Blood*).

Erythrocythæmia.—An increase in the number of red blood corpuscles, such as is supposed to occur in hæmophilia.

Erythrocytorrhexis.—The breaking up of red corpuscles and the escape from them of round granules; plasmorrhexis.

Erythrocytoschisis.—The degeneration of the red blood corpuscles and the formation from them of cells like the blood-platelets; plasmoschisis.

Erythrocytosis.—The occurrence of red cells of the fœtal type (*e.g.* nucleated) in the blood.

Erythrodextrin. — A dextrin, formed by the action of the saliva on starch, and giving a brown colour with iodine (hence its name). *See* Physiology, Food and Digestion (*Salivary Digestion*).

Erythrol Nitras.—A non-official drug, given in doses of ½ to 1 grain, and having the same action as the nitrites (*e.g.* nitrite of amyl).

Erythromelalgia. — A morbid condition, resembling Raynaud's disease, in which paroxysmal pain, redness, and swelling occur, more particularly in the feet. *See* Raynaud's Disease (*Erythromelalgia*); Dermatitis Traumatica et Venenata (*Eruption due to metol*); Hysteria (*Symptoms, Disorders of Circulation*); Nails, Affections of (*Diseases of the Nervous System*); Nerves, Multiple Peripheral Neuritis (*Arsenical Neuritis, Symptomatology*).

Erythrophlœum.—A non-official drug, known also as Casca or Sassy bark, containing an alkaloid (*Erythrophlocina*), and given in the form of a Tinctura Erythrophlœi (dose, 5 to 10 m.) as a cardiac tonic. *See* Alkaloids (*Vegetable*).

Erythropsia.—A disorder of vision, in which everything seems to be of a red colour.

Erythroxylon Coca. — *See* Coca; Cocaine.

"Esau."—A popular name given to hairy individuals; a case of hypertrichosis congenita, polytrichia, hirsuties, or dasytes.

Esbach's Method.—The quantitative estimation of the albumin in urine by means of citric and picric acid. *See* Urine, Pathological Changes in (*Albuminuria*, *Tests*).

Eschar.—A slough, produced, for instance, by a caustic (also called an *Escharotic*), or as a trophic change in some diseases (*e.g.* hemiplegia). Well-known escharotics are silver nitrate, strong nitric acid, arsenious acid, and caustic potash.

Eserine.—The alkaloid of Physostigmatis Semina or Calabar Beans. *See* Physostigmatis Semina; Alkaloids (*Vegetable*); Cornea (*Ulcerative Keratitis, Treatment, Eserine*); Glaucoma (*Treatment, Myotics*); Pharmacology; Toxicology (*Alkaloids, Physostigmine*).

Esmarch Bandage. *See* Aneurysm (*Treatment, Compression*); Bandages, Varieties (*Triangular*); Hæmorrhage (*Local Treatment, Elastic Constriction*).

Esodic.—Afferent (*e.g.* nerves).

Esophoria.—The condition in which one eye tends to deviate inwards towards the other, as contrasted with exophoria, in which the deviation is outwards. *See* Ocular Muscles, Affections of (*Abnormal Position*); Strabismus (*Strabismus Convergens, Esophoria*).

Esoteric.—The name given to conditions or bodies developing within the organism, in contrast with *Exoteric.*

Essences. — Essences or Essentiæ are strong solutions of volatile oils in rectified spirit; they may be given (on loaf sugar) as carminatives; the *essentia anisi* and the *essentia menthæ piperitæ* are not in the Pharmacopœia of 1898. *See* Prescribing.

Essential Oils.—Volatile or essential oils are usually mixtures of liquid hydrocarbons (elæoptens) with solid hydrocarbons (stearoptens) and sometimes with resins. Many essential oils are used medicinally, generally as solutions in alcohol; among these may be named oil of anise, of dill, of peppermint, of chamomile, of bitter orange, of lemon, of cardamoms, of cajuput, etc. *See* Prescribing; also under the various drugs (*e.g.* Anethi Fructus, Anisi Fructus, etc.).

Esters. — Esters, compound ethers, or ethereal salts, are salts of alcoholic radicles (the *ethers* being oxides of alcoholic radicles); they are formed from the paraffins, the hydrogen being displaced by compound acid radicles; examples are found in ethyl nitrite ($C_2H_5NO_2$), ethyl sulphate ($(C_2H_5)_2SO_4$), and ethyl acetate ($C_2H_5C_2H_3O_2$).

Esthiomene. — Tuberculosis of the vulva or lupus vulvæ; as originally defined by Huguier it probably included other diseases. *See* Vulva, Diseases of (*Cutaneous, Tuberculosis*).

Estlander's Operation.—Thoracoplasty. *See* Pleura, Affections of, Surgical (*Thorax-Resection*).

"Estoral." — A boric-menthol ester (*Zimmer and Co.*), recommended in various forms of rhinitis.

Ethane.—A paraffin, C_2H_6, regarded as derived from two methane molecules (C_2H_8) by taking away a hydrogen atom from each; dimethyl; ethyl hydride. *See* Physiology, Organic Chemistry.

Ether. *See* Anæsthesia and Anæsthetics (*Ether*); Morphinomania and other allied Drug Habits (*Ether Drinking*); Temperature (*Alterations, Depression, Etiology*).

Ethics, Medical.—The science and practice of the maintenance of correct relationships between medical practitioners in connection with their patients; medical etiquette. It is founded on two great laws—the first is to do to your brother practitioner as you would have him to do to you; the second is to secure the safe, certain, and, if possible, speedy recovery of the patient. The former is a perfectly definite indication, although, unless a man be free from pride and little afflicted by the love of money, it may be difficult to carry through; the second is also a definite law, although much skill and experience may be needed for its accomplishment, but it is when we attempt to combine the two laws in any given case that the real difficulties of medical etiquette begin.

Ethmocephalus. — A teratological type in which the orbits are approximated; the nose is represented by a proboscis, and there are usually other facial defects; a form of cebocephalus (*q.v.*).

Ethmoidal Cells. *See* Nose, Accessory Sinuses, Inflammation.

Ethmoiditis. — Inflammation in the ethmoid bone and in its neighbourhood.

Ethnology. *See* Anthropology; Anthropometry; Physiognomy. Ethnology is the

science which deals with the races of mankind, more especially in their relations to each other, and *ethnography* may be simply defined as descriptive ethnology. It is now generally admitted that the four fundamental groups into which the various races can be classified were marked off and established as early as the Neolithic or New Stone Age. These four groups are the Negro, the Mongolian, the American, and the Caucasic. Some of the ethnical criteria or features and characters which distinguish the primary groups from each other are of special interest to medical men; such are the anatomical peculiarities of the skull, the face, the jaws, the skin, the hair, the nose, and the eyes. The *negro* race, for instance, in its two subdivisions (African and Oceanic) is characterised by the possession of black woolly hair which appears flat in transverse section, of a dolichocephalic skull, of a prognathous lower jaw, of a broad, flat nose, of a black or dark brown skin which emits a peculiar odour, of large, black, round eyes with a yellowish cornea, and of a stature which is above the average (*e.g.* 5 ft. 10 in.). With regard, however, to the last-named character, it has to be noted that negro dwarf races, such as the Akkas of Central Africa, the Bushmen, the Batwas, and the Andamanese, are well known; in all these races the average height is less than 5 feet. The *Mongolic race* includes the Mongols, Tartars, Finns, Laps, Magyars, Koreans, Japanese, Tibetans, Burmese, Chinese, Malays, Formosans, and others. These are nearly all of markedly short stature (*e.g.* 5 ft. 4 in.); they have a yellowish or yellowish-brown skin; their hair is coarse and lank, usually black, and round in transverse section; they have brachycephalic skulls and mesognathous or orthognathous jaws; the nose is small and often snub; and the eyes are small, black, and oblique, and possess an epicanthic fold (*q.v.*). To the *American* group belong the Eskimos, the various tribes of North American Indians, the Aztecs and other Mexican tribes, the Incas and South American tribes, and the Patagonians and Fuegians. The skin in these American races is yellowish brown or coppery; the hair is long and coarse, and almost round in transverse section; the skull may be dolichocephalic or brachycephalic; the stature is above the mean (5 ft. 8 in. or more); the jaw is mesognathous, and the nose large and arched; and the eyes are small, round, and straight. The *Caucasic*, or fourth group of races, includes a large number of varieties. There is first the Hamito-Semitic, containing the Hamitic section (Egyptians, Somalis, Berbers, Basques, Early Greeks, Etrurians, etc.), and the Semitic (Arabs, Assyrians, Syrians, Israelites, Moabites, Phœnicians, etc.). There is, second, the Indo-European, with its Hindu, Dravidic, Iranian (Persian, Afghan, Armenian, Kurdic), Hellenic, Italic, Celtic, Lettic, Slavonic (Russian, Bohemian, Polish, Bulgarian, Servian, Montenegrin), and Teutonic peoples. There is, third, the Caucasian group properly so called, including Georgians, Circassians, and Avars. There is, fourth, the Indonesian, including chiefly the natives of certain islands in the Pacific (Samoan, Tongan, Maori, Hawaian); and, finally, in a fifth subdivision, are the Ainus. The Caucasic peoples have, as a rule, a white skin; the hair is long, straight or wavy, flaxen, red, brown, or black, and oval in transverse section; the skull may be dolichocephalic or brachycephalic; the jaw is orthognathous; the nose is large, straight or arched; the stature is medium (5 ft. 4 in. to 6 ft.); and the eyes are moderately large, straight, and of various shades of colour (blue, black, brown, grey). Some attempts have been made towards a study of Comparative Nosology, and it has been thought that certain races are more susceptible to certain diseases and immune against others. It would appear, for instance, that hæmophilia is rare in the negro and pulmonary diseases in the Arab; the Jews also seem to have special tendencies. Ethnological pathology is still, however, largely an unworked subject.

Ethyl.—The hypothetical radicle (C_2H_5), which enters into the composition of ethyl alcohol (C_2H_6O), ethylamine (NC_2H_7), etc.

Ethyl Chloride. *See* ANÆSTHETICS, ETHYL CHLORIDE.

Ethylene.—Olefiant gas (C_2H_4), derived from ethane (C_2H_6) by the loss of two hydrogen atoms; it is usually got from ethyl alcohol (C_2H_6O) by the action of concentrated sulphuric acid at a high temperature; it combines with chlorine, bromine, and iodine to form ethylene chloride ($C_2H_4Cl_2$), bromide ($C_2H_4Br_2$), and iodide ($C_2H_4I_2$); the chloride has been used as an anæsthetic and the bromide in epilepsy.

Ethyl Nitrite.—There is an official solution of ethyl nitrite (the *liquor ethyl nitritis*), a yellow, inflammable liquid, with an odour of apples; it is given, in doses of 20 to 60 m., in the same kind of cases (angina pectoris, dyspnœa due to heart disease, etc.) as amyl nitrite and nitro-glycerin.

Eucaine. *See* ANÆSTHETICS (*Local Anæsthesia*); COCAINE (*Eucaine*); NOSE, LOCAL ANÆSTHETICS (*Eucaine*); TEETH (*Tooth Extraction, Eucaine*).

Eucalyptus Gum. — Red gum or eucalyptus gum is obtained from the bark of various Australian species of eucalyptus; it contains kino-tannic acid, catechin or catechinic acid, and pyrocatechin or catechol; it is an astringent, and is used in diarrhœa and dysentery (dose, 2 to 5 grains), and, as the *Trochiscus*

Eucalypti Gummi, in relaxed throats; it is also employed as a decoction, a liquid extract, and in the form of suppositories and vaginal and rectal douches.

Eucalyptus Oil.—*Oleum Eucalypti* is obtained from several Australian species of Eucalyptus (including the *E. globulus*, or blue gum tree); it contains eucalyptol (a volatile oil), a resin, tannin, and cineol ($C_{10}H_{18}O$); there is an official preparation (*Unguentum Eucalypti*), and the oil itself is given in doses of ½ to 3 m.; internally it acts as an antiseptic, an anti-periodic, an expectorant, and a carminative, and it has been used in bronchitis, coryza, pyelitis, and malaria; externally it is an antiseptic, and may be used in the form of gauze, as a solution, or as an ointment. "Mulyptol" is a proprietary preparation; it is said to be a eucalyptus oil of great purity and high therapeutic and oxygenating properties; it is given in doses of 2 to 3 m. on loaf sugar or in emulsion.

Euchinin. *See* EUQUININE.

Euchromatopsia. — Normal colour vision, as opposed to colour-blindness.

Eudermol.—A proprietary preparation used in certain skin diseases; said to be nicotine salicylate.

Eudoxin. — A proprietary preparation, said to be a bismuth salt of nosophen; recommended, in doses of 5 to 8 grains, in cases of intestinal irritation, such as enteritis.

Euformal.—A proprietary preparation containing formaldehyde and other substances, used as an antiseptic.

Eugenics.—The science of well-begetting, good breeding, or the procreation of the fittest, as regards the human race. Francis Galton seems to have been the first to use the word in this sense. In the Huxley Memorial Lecture (1901) he proposed a system of dowries to make possible the early marriage of girls of a favoured stock (as regards health). Before, however, much can be done either in the way of favouring the marriage of the fit, or preventing the marriage (or, at least, the fruitful marriage) of the unfit, our knowledge of antenatal hygiene and of antenatal pathology must be greatly widened and remarkably systematised and defined. At the present time we do not know the laws which go to the making of a normal, healthy infant who shall be free from hereditary taints, for we not infrequently are surprised by the birth of a healthy baby to parents who are not of the best stock, and disappointed by the procreation of a deformed or diseased infant by apparently healthy parents.

Eugenol. — Eugenol, or eugenic acid, ($C_{10}H_{12}O_2$) is a phenol-like substance contained in the oil of cloves, the oil of cinnamon, and the oil of pimento; it has been used as an antiseptic and antipyretic.

Euguform. — Acetilated diguaiacol of methylene, recommended to allay pain and itching in some skin diseases.

Euhemerism.—The theory according to which "myths are regarded as traditional accounts of real incidents in human history"; by this theory the teratological appearances of many of the heathen gods and goddesses are to be explained by the birth in early times of malformed fœtuses (*e.g.* Cyclops or Polyphemus and the cyclopic infant).

Eumydrin. — A powder obtained from atropine, which acts as a mydriatic, and is said to be less poisonous than atropine.

Eunatrol.—A proprietary preparation of oleate of sodium, given in doses of 2 to 4 grains, as a cholagogue, and said to be beneficial in cases of colic from gall-stones.

Euonymin. *See also* CHOLAGOGUES; PHARMACOLOGY; etc. — *Extractum Euonymi Siccum*, derived from the bark of Euonymus atropurpureus. *Dose*—1-2 grs. It contains a bitter principle, some resin, and a small amount of fixed oil. Although Euonymin possesses some action as a bitter stomachic, it is as a purgative that it is almost exclusively employed. It is said to increase the flow of bile, and is specially recommended in cases of biliousness associated with constipation. It is usually prescribed in combination with calomel or other cholagogue.

Eupareunia.—The condition in which sexual congress occurs without pain, as opposed to dyspareunia (*q.v.*).

Eupepsia.—Normal digestion, as opposed to dyspepsia (*q.v.*).

Euphthalmine. — An alkaloid ($C_{16}H_{25}NO_3$), the hydrochloride of which acts as a mydriatic; its use has been observed to be followed by acute glaucoma (H. W. Ring, *Med. News*, vol. lxxxiii. p. 82, 1903).

Eupnœa.—Normal respiration, neither dyspnœa nor apnœa. *See* ASPHYXIA (*Definition*).

Euquinine. — Ethyl-carbonic ester of quinine; it is tasteless, and is used, as the hydrochloride or the tannate, in malaria, pertussis, etc.; it is only slightly soluble in water.

Europhen.—A non-official substance, acting like iodoform as an antiseptic; it contains 28 per cent of iodine.

Eurygnathism. — The condition in which the upper jaw is broad.

Eustachian Tube. *See* EAR, EXAMINATION OF (*Anatomy, Examination by Catheter, etc.*); EAR, MIDDLE, CHRONIC NON-SUPPURATIVE DISEASE (*Chronic Eustachian Catarrh*); GLOSSO-PHARYNGEAL NERVE; NOSE, EXAMINATION OF (*Posterior Rhinoscopy*); PHYSIOLOGY, SENSES, HEARING (*Middle Ear*).

Eustrongylus. *See* PARASITES (*Nematodes, Strongylidæ*).

Euthanasia.—A painless death, or, more particularly, the result of the measures which can be adopted to prevent the suffering (mental and bodily) which often precedes death; such suffering may be due to pain (as in cancer), or breathlessness (as in various forms of asthma and pulmonary and cardiac troubles), or cough (as in phthisis), or abdominal distension (as in ascites).

Euthermic Stove. — A means of warming and ventilation combined, in which pipes containing the fresh air pass into the room through a gas stove.

Eutocia.—Easy and uncomplicated labour (*see* LABOUR, PHYSIOLOGY OF), in contrast with *dystocia*, or painful and difficult parturition.

Eutrophia.—A well-nourished condition of body; opposed to *dystrophia*.

Evacuants.—Purgative medicines, or, more generally, all drugs which cause the evacuation of any secretion or excretion from the body (*e.g.* emetics, diuretics, diaphoretics, and expectorants).

Eventration.—Protrusion of some of the abdominal contents through a space (*e.g.* between the recti muscles) in the abdominal parietes; ventral hernia.

Evian. *See* MINERAL WATERS (*Alkaline, Simple*).

Evian-les-Bains. *See* BALNEOLOGY (*France, Alkaline*).

Evidence. *See* MEDICINE, FORENSIC (*Examinations, Circumstantial Evidence*).

Evil.—A disease; now almost obsolete in this sense; the "falling evil" was epilepsy, the "king's evil" was scrofula, the "yellow evil" was jaundice, the "foul evil" was the pox, and the "Aleppo evil" was the furunculus orientalis.

Evisceration. — The removal of any organ (*e.g.* the eyeball) or organs (*e.g.* the abdominal viscera) from the cavity in which they normally lie; a form of embryotomy in difficult labour. *See* LABOUR, OPERATIONS (*Embryotomy*).

Evolution.—A form of spontaneous delivery which sometimes occurs in cases of transverse presentation in labour. *See* LABOUR, DIAGNOSIS AND MECHANISM (*Transverse Lies, Spontaneous Delivery*). The gradual development of an organ or organism (*see* EMBRYOLOGY), or of a species from a simpler to a more complicated state (*see* HEREDITY; DARWINIANISM).

Ewald's Test Meal. — A slice of bread and two cups of weak tea without milk or sugar are taken on a fasting stomach, and withdrawn through a tube after the lapse of an hour. The digestive power of the stomach is determined by chemical examination of the material so obtained.

Exalgin. *See also* ANALGESICS AND ANODYNES.—*Methyl-acetanilide*; soluble freely in alcohol, and sparingly in water. *Dose*—½-3 grs. It is a very useful analgesic, but must be given with caution, as it possesses a toxic action resembling that of acetanilide when too large doses are employed. It is indicated in all forms of neuralgia, in the pains of locomotor ataxia, in painful skin diseases, and in some forms of cardiac pain. It has proved especially helpful as a substitute for opium in relieving pain due to inoperable malignant disease. Although certainly not so popular now as it was a few years ago, exalgin is well worthy of a trial when other analgesic remedies fail.

Exaltation. — That stage or type of mental disease in which the patient is possessed by ideas of his well-being, wealth, grandeur, power, and importance; it is met with in many forms of insanity, but is specially prominent and often referred to in general paralysis. *See* INSANITY, NATURE AND SYMPTOMS (*Delusional Insanity, Monomania of Exaltation*).

Examination. *See* GYNECOLOGY, DIAGNOSIS IN; POST-MORTEM METHODS; SKIN, PARASITES (*Method of Examination*); FOOD (*Inspection*); SCHOOL CHILDREN; etc.

Exanthemata.—The eruptive fevers, such as scarlet fever, small-pox, measles, varicella, "fourth disease," etc. *See* under these maladies.

Excerebration.—The removal of the brain in order to diminish the size of the fœtal head in labour and so to expedite delivery. *See* LABOUR, OPERATIONS (*Embryotomy*).

Excision. *See* ANEURYSM (*Treatment, Local, Excision*); ANKLE-JOINT, REGIONS OF, OPERATIONS (*Excision*); ELBOW-JOINT, INJURIES AND DISEASES (*Operations, Excision*); HIP-JOINT, DISEASES OF (*Operative Treatment, Excision*); KNEE-JOINT, DISEASES OF (*Treatment, Excision*); MAMMARY GLAND, DISEASES OF (*Carcinomata, Treatment*); SHOULDER, DISEASES AND INJURIES

of (*Operations, excision of head of humerus*); Tongue (*Carcinoma, Operative Treatment*); Wrist-Joint Diseases (*Tuberculosis, Operative Procedures*).

Excitants.—Drugs or applications which increase the functional activity of any organ of the body; stimulants. *See* Pharmacology.

Excoriation.—An abrasion or erosion of the skin or of a mucous membrane.

Excrescence.—A tumour attached to and growing from the skin or a mucous surface, *e.g.* the cauliflower excrescence of the cervix uteri (cervical epithelioma), or warty excrescences of the vulva (condylomata).

Excreta. *See* Fæces.

Excretion. *See* Physiology, Tissues (*Effect of Muscular Work*); Physiology, Respiration, Excretion by the Kidneys and by the Skin.

Exencephalus. — The teratological type in which the posterior part of the cranial vault is defective, and the cranial contents lie exposed on the nape of the neck. *See* Teratology; Cheek, Fissure of (*Complications*).

Exenteration.—Evisceration (*q.v.*). *See* Labour, Operations (*Embryotomy, in Trunk Presentations*).

Exercise. *See* Alcoholism (*Treatment, Personal, General*); Heart, Myocardium and Endocardium (*Treatment, Rest, and Exercise*); Pregnancy, Management (*Exercise*); Schott Treatment.

Exfoliation.—The separation of dead tissue, epidermic or osseous, or (occasionally) of mucous membrane (*e.g.* of the bladder in cystitis).

Exhumation.—The digging up of a corpse which has been buried for some time in order to investigate the cause of death, especially in cases of suspected poisoning.

Exitus Lethalis.—The fatal termination, *e.g.* of an illness.

Exocardia. — A teratological state in which the heart lies external to the chest, and there is a fissure of the sternum; it may be associated with diaphragmatic hernia.

Exodin.—A yellow, tasteless powder, related to emodin and purgatin; it is derived synthetically from oxyanthraquinon; it is used, in doses of 7 to 15 grains, as a mild purgative.

Exohysteropexy.—The fixation of the uterus to the anterior abdominal wall (*e.g.* in cases of prolapse) by burying part of the organ between the layers of the wall.

Exomphalos.—Defect or fissure of the anterior abdominal wall, allowing protrusion or exposure of the viscera; abdominal or ventral hernia; gastroschisis. *See* Fœtus and Ovum, Development (*Umbilical Cord*); Labour, Faults in the Passenger (*Monstrosities, Exomphalos*); Pregnancy, Ovum and Decidua (*Affections of the Amnion, Inflammation*); Teratology.

Exophoria. *See* Esophoria; Ocular Muscles, Affections of (*Abnormal Position*); Strabismus (*Classification, Strabismus Divergens*); Syphilis (*Tertiary, Eye and its Appendages*).

Exophthalmic Goitre. *See* Thyroid Gland, Medical (*Exophthalmic Goitre*); *see also* Adrenal Glands, Addison's Disease (*Pigmentation in Exophthalmic Goitre*; Aneurysm (*Diagnosis*); Cornea (*Ulceration from Exposure*); Hæmatoporphyrinuria; Pulse (*Frequency, Increase in*); Skin, Pigmentary Affections (*Exophthalmic Goitre*).

Exophthalmos. *See* Aneurysm (*Arterio-venous Intercommunications, Pulsating Exophthalmos*); Brain, Affections of Blood-Vessels (*Thrombosis, Morbid Anatomy*); Brain, Cerebellum, Affections of (*Symptomatology, Ocular Nerves*); Eye, Clinical Examination of; Leontiasis Ossea (*Localised Hyperostosis*); Lungs, Vascular Disorders (*Embolism, Clinical Features*); Nose, Accessory Sinuses, Inflammation (*Acute Inflammation, Ethmoidal Cells*); Nose, Nasal Neuroses (*Grave's Disease*); Orbit, Diseases of (*Pulsating and Intermittent Exophthalmos*); Orbit, Diseases of (*Thrombosis of Cavernous Sinus*); Rheumatism, Rheumatoid Arthritis (*Clinical Characters*); Scurvy, Infantile (*Diagnosis*); Thyroid Gland, Medical (*Exophthalmic Goitre*).

Exosplenopexy. — An operation in which the spleen is fixed into an incision made in the abdominal wall.

Exostosis. *See* Ear, External, Diseases of (*Stenosis of Meatus from Exostoses*); Labour, Precipitate and Prolonged (*Faults in the Passages, Tumours of the Pelvic Bones*); Orbit, Diseases of (*Tumours*); Teeth (*Exostosis*).

Expectant Treatment. — The management of an illness by non-interference, especially operative, until, at least, symptoms very clearly call for more active measures.

Expectation of Life.—The probabilities of life and death at any age as brought out by statistics ("life tables"); mean after-lifetime; in England the expectation of life at birth is 43·66 years for the male and 47·18 years for the female. *See* Vital Statistics (*Life Tables*).

Expectorants.

See also Acids; Antimony; Benzoic Acid; Cough; Expectoration; Ipecacuanha; Jaborandi; Pharmacology; Senega; Squills; Sulphur; Turpentine; etc.

These are remedies which promote the removal of abnormal secretion from the respiratory tract. As there is no class of remedy more abused in practice owing to defective appreciation of the exact pathological condition in different diseases, and at different stages of the same disease, it is advisable to specially indicate the important pathological and clinical points that call for consideration. An expectorant may act primarily by stimulating the pulmonary circulation, thus promoting an increased secretion; at the same time this action may be assisted, as in the case of ammonia, by its general stimulating effect on the heart, the central nervous system, and the nerve-endings in the respiratory tract. The preparations of ammonia and especially ammonia carbonate may be taken to represent the group of so-called *stimulating expectorants*. The alkalies, and very specially iodide of potassium, ipecacuanha, antimony and antimonial salts, and lobelia, are examples of expectorants whose action is associated with a depression of the general circulation and a corresponding tendency to diminish bronchial secretion. Whereas the former are specially useful in the later stages of catarrh associated with diminished expulsive power in the bronchi, the latter are more serviceable in the earlier stages when there is congestion of the respiratory passages and scanty expectoration. In another class of case so-called *sedative expectorant* treatment is adopted, for example, where coughing is persistent and severe, and out of all proportion to the amount of secretion, hydrocyanic acid, compound tincture of camphor, codeine, or morphine may be indicated, the two latter, however, only under exceptional circumstances. The volatile antiseptics, especially eucalyptus, terebene, olei pini sylvestris, turpentine, and the like, are of most service in those cases where purulent expectoration is very pronounced. These volatile substances are, however, of greater service in acute catarrh when used as inhalations in appropriate strengths, *e.g.* Ol. pini sylvestris ʒss.-j. to a pint of boiling water, although it must be admitted that the hot moist air alone may suffice to give marked relief in those cases where the inhalation plan of treatment is advisable.

General tonic remedies are of great importance. These include particularly cod-liver oil, arsenic, nux vomica, and iron, and a judicious use of one or other of these remedies, either in very young or in old subjects, frequently proves successful when ordinary expectorant remedies have ceased to be beneficial. The severe early morning coughing in chronic bronchitis is favourably influenced by the use of hot fluids at night, *e.g.* hot toddy, gruel, or milk and water, assisted by administration of pot. iodide (gr. x.) in very dilute form twice or thrice daily.

Occasionally a persistent cough, the result of previous local respiratory trouble, may be got rid of by mild stomachic treatment, *e.g.* small doses of rhubarb and soda.

Before recommending any remedy for the cure of a cough or removal of expectoration in chronic conditions, very careful consideration should be called to the following points:—

(*a*) To gauge the condition of the bronchial mucous membrane from the history of the patient, the amount of secretion, and the nature, time, and severity of the coughing.

(*b*) To corroborate this information obtained by careful physical examination, and specially the amount of moist and dry sounds, emphysema, etc. In this examination the bases of the lungs should be specially examined.

(*c*) The condition of the heart and circulation, and notably the right heart.

(*d*) The *personnel* of each individual patient.

Careful consideration of these points will usually suffice to indicate the special line of treatment to be followed. It will sometimes be found that the use of an expectorant remedy or combination of remedies is the least important point in treatment. Too much attention cannot be paid to the beneficial influence of a change of air, to the thorough and constant invigoration of the functions of the skin and other excretory organs, by the use of suitable clothing, baths, exercises, and the like.

Expectoration.

See also Fluids, Examination of Pathological; Lung, Tuberculosis of (*Clinical Features, Expectoration*); Lungs, Gangrene (*Symptoms*); Micro-organisms.

Under the term sputum or expectoration we include all substances expelled by coughing from the respiratory tract below and including the larynx. The sputum is, of course, almost invariably contaminated by nasal secretion and saliva; these, however, will not be considered here.

1. General Characters.—(1) *Amount.*—This varies greatly according to the nature and extent of the disease; in some cases there may be

only a few cubic centimetres in the day, in others a pint or more. It is important for several reasons to note the quantity: it gives a clue to the progress of the disease; if excessive it may be a serious drain on the system, since as much as 5 per cent of the total nitrogen excreted is sometimes eliminated in the sputum; finally, an abundant expectoration is often an indication for remedies calculated to diminish its amount. Children up to the age of 6 or 7 seldom expectorate. Sputum for examination, however, may sometimes be obtained from the pharynx on a swab. The aged or enfeebled may not expectorate—often a most unfavourable sign, both as indicating great prostration and causing mechanical embarrassment of the respiration.

(2) *Colour.*—In its simplest form—an increase of the normal bronchial secretion—the sputum consists of clear mucus. When the catarrh is more marked the addition of cells gives a whitish yellow, or (from admixture of particles of carbon, etc.) a greyish white colour. With the increase of cells, as the purulent condition is reached, the spit becomes yellower or greenish. The exact cause of the bright yellow or green tinge of certain purulent sputa is unknown; in some cases it may be due to altered hæmoglobin, in others to bile-pigment, and in the others to the action of chromogenic bacteria. The most typical examples of purulent sputa are found where an empyema or hepatic abscess perforates the lung. In the latter case, if the abscess be due to the amœba coli, the sputum is of a brick-red colour.

Another variety of clear translucent sputum (not infrequently, however, slightly blood-stained) occurs in pulmonary œdema, where large quantities of watery fluid—blood serum—may be brought up.

In hæmoptysis blood may be merely streaked through the sputum, it may be intimately mixed with it, or the expectoration may consist almost wholly of blood. If the blood be well aerated the tint is bright red; otherwise it is darker and venous. The colour, however, is frequently altered; the rusty sputum of pneumonia is well known, in gangrene of the lungs the sputum is often prune-juice coloured, and in other cases where the sputum has been retained for a long time, hæmatoidin may develop and give a chocolate colour. In carcinoma of the lung the sputum sometimes resembles red-currant jelly; in chloroma, and in some forms of sarcoma, it is bright green. Black sputum occurs in anthracosis, while in siderosis it may be ochre-colour, and give the iron reaction. If these alterations are due merely to inhaled particles they cease when the patient desists from his occupation. Their persistence after this is indicative of a destructive (generally tuberculous) lesion of the affected lung.

(3) *Consistence.*—The most watery sputum is that of œdema of the lungs; the most viscid are those consisting largely of mucin, as in the early stage of acute bronchitis. Purulent and muco-purulent sputa lie midway between these extremes. The great viscosity of pneumonia expectoration is probably due, not to mucin, but to the presence of nuclein. The amount of air in the sputum depends partly on its consistence and partly on its origin. *Cœteris paribus*, the air-content is greater in sputa arising from the finer bronchi.

(4) *Odour.*—The expectoration is usually odourless, or, if purulent, has a slightly mawkish smell. If it be retained in the lungs for any time it may become extremely offensive, while very fœtid sputa occur in gangrene of the lung and bronchiectasis, in the latter of which especially the odour is most acrid and penetrating. When tyrosin is present the sputum is said to have a cheesy smell. Such aromatic drugs as paraldehyde, turpentine, etc., may communicate their odour to the expectoration.

(5) *Form.*—The more viscid sputa may retain a characteristic shape after expectoration. The well-known nummular spit of phthisis and the less common fibrinous casts in plastic bronchitis are examples of this. Nummules consist of collections of muco-pus, usually about the size of a bean, which tend to remain separate, and float or sink in water according to the amount of air they contain. Another characteristic sputum is one containing small, yellowish, cheesy masses. These are found in three conditions—phthisis, bronchiectasis (Dittrich's plugs), and where the caseous masses from the tonsillar crypts contaminate the sputum. Dittrich's plugs and tonsillar concretions are fœtid, and consist of degenerated epithelium, bacteria, crystals, detritus, etc. In phthisis the masses are odourless, and contain numbers of tubercle bacilli.

When the expectoration is allowed to stand in a conical glass it frequently separates into three layers—a lower one of cells and the denser constituents generally, a middle, liquid portion, and an upper, frothy layer.

(6) *Composition.*—There are four main varieties of sputum—mucous, purulent, serous, and sanguineous. Commonly, however, an expectoration combines one or more of these characters—sero-sanguineous, muco-purulent, etc. Sputa due to catarrh of the respiratory tract are mucous or muco-purulent; those due to œdema, serous. Transient copious serous expectoration may follow paracentesis thoracis. It is simply due to passing œdema of the lung. There is some doubt as to whether mucin is present to any considerable extent in the sputum of croupous pneumonia. When the catarrh is severe the cellular elements may increase until the sputum consists almost entirely of pus. An expectoration of pus occurs in pulmonary abscess, perforating empyema, etc. Blood may be found

in small amount after severe coughing from rupture of some of the capillaries in the upper air-passages; it is then of no significance. One of the most common causes of hæmorrhage is a destructive change—almost invariably tubercular—in the pulmonary alveoli. Again, bleeding may be due to the bursting of a large vessel into a cavity; in such cases pure blood is brought up, often in great quantity. A slight admixture of blood is very common in serous sputa from rupture of the engorged pulmonary capillaries. A somewhat characteristic muco-sanguineous sputum may be caused by the "weeping" of a thoracic aneurysm which is eroding the wall of a bronchus; this often presages rupture of the sac.

Little of practical worth has yet accrued from the *chemical examination* of the sputum. The chief albuminous constituents are mucin and nuclein, while in serous sputa we have serum-albumin, and in purulent sputa peptone. Fatty acids, glycogen, ferments, and various inorganic salts have also been found, but none of them have any particular clinical significance.

(7) *Specific Gravity and Reaction.*—The specific gravity of the sputum lies between 1004 and 1037; it is of no importance. The reaction is always alkaline.

II. The Sputum in various Diseases.—*Bronchitis.*—In acute bronchitis the sputum is at first scanty, viscid, and frothy, consisting chiefly of mucus; later it becomes more abundant and muco-purulent from the admixture of cells. Most of the cells are leucocytes, and in certain cases very many of these are eosinophilous. It has been proposed to call such cases (which have a subacute course, with relapses) eosinophilic bronchitis, but they do not appear to have such definite clinical features as to merit a special name.

In the capillary bronchitis of childhood and age expectoration is commonly absent.

In chronic bronchitis the expectoration may be very slight; usually, however, it is fairly abundant and muco-purulent. Sometimes it is so copious as to deserve the name bronchorrhœa, which may be mucoid or purulent. The sputum is usually odourless unless putrefactive changes ensue. It is generally most abundant in the morning, and is often expelled in large mouthfuls. It contains leucocytes, epithelium, and sometimes fatty acid crystals. The last named, as well as moulds and yeasts, point to retention of the sputum in the bronchi. In dry chronic bronchitis scanty, tough pellets of mucus, sometimes containing spirals and Charcot-Leyden crystals, are expectorated. Should bronchitis complicate another malady the sputum may derive from the blood substances—*e.g.* bile, sugar, urea—whose presence therein is due to the primary disease.

Bronchiectasis and Putrid Bronchitis.—In the former the sputum may be odourless; commonly, however, it is extremely fœtid. The expectoration is darker than in bronchitis; it has a somewhat oily consistence, and on standing separates into three layers. It consists chiefly of mucus and pus cells, but may also contain Dittrich's plugs, various organisms (to one of which, possibly, the odour may be due), crystals of hæmatoidin, cholesterin, and fatty acids, Charcot-Leyden crystals, and yellow elastic tissue. Dittrich's plugs are caseous masses originating in the smaller bronchi, and composed of debris, fat globules, crystals, putrefactive organisms, cercomonas, leptothrix, sarcinæ, etc. Hæmoptysis may occur in bronchiectasis.

Plastic Bronchitis.—Large fibrinous casts may be expectorated, which when floated out in water show the ramifications of the bronchi down to their finest terminations. In other cases only small casts of the bronchioles are brought up, though often in considerable numbers. Curschmann's spirals, eosinophile cells, and Charcot-Leyden crystals may accompany the casts.

Asthma.—A mucous expectoration, with more or less admixture of cells, is present only towards the end of the paroxysm. Three special constituents require mention: Curschmann's spirals, which are more common in this disease than in any other, eosinophile leucocytes, and Charcot-Leyden crystals.

Croupous Pneumonia.—In the early stage the viscosity of the sputum is sometimes so great that the spittoon can be inverted without its contents falling out. At this time the sputum may be clear, or but slightly yellow, but the typical rusty tint soon develops. In uncomplicated cases the sputum is never very abundant; it persists for a day or two after the crisis. By floating it out in water casts of the bronchioles can sometimes be seen. It contains a few leucocytes and a fair number of red cells, many of the latter, however, having yielded up their pigment to the ground substance of the expectoration. The most important constituent is the exciting organism—usually the pneumococcus, but sometimes the pneumobacillus or some other microbe.

In almost no disease is the daily examination of the sputum so important as in pneumonia. Instead of being rusty, it may be greenish-yellow, chocolate, or prune-juice coloured. Should it suddenly become abundant and serous, the onset of œdema will be suggested. The expectoration of large quantities of dark-brown, frothy sputum is a bad sign; it is frequently due to localised necroses of lung tissue, the areas being too small to yield recognisable physical signs. Other unfavourable terminations may be shown by a copious purulent spit, indicating (in the absence of bronchiectasis) abscess-formation, or by the occurrence of the fœtid expectoration of pulmonary gangrene.

Pulmonary Tuberculosis.—Owing to the extreme variability of the course and morbid

anatomy of this disease, the naked-eye appearances of the sputum vary so greatly as to be of little diagnostic value. The amount and nature are, however, a rough guide to the activity of the disease and the condition of the patient.

In acute miliary tuberculosis the sputum is scanty, and resembles that of the earlier stage of acute bronchitis; it may, indeed, be absent. In ordinary chronic phthisis we usually find a muco-purulent sputum with occasional hæmoptysis. Perhaps the most characteristic feature of the sputum is its heterogeneity. Some parts are but slightly purulent and consist chiefly of mucus, others are more densely yellow and may form nummules, while here and there little cheesy masses, or, more rarely, calcareous granules are seen. Though suggestive of excavation, nummules are not pathognomonic of it, since they may arise in the bronchi. Hæmoptysis may be so slight as only to tinge one or two spits, it may occur as a continuous oozing, or may be severe and rapidly fatal. The microscopic constituents whose importance causes them to overshadow all others are yellow elastic tissue and tubercle bacilli.

Gangrene of the Lung.—The fœtor is extreme, but less pungent than in bronchiectasis. The sputum is abundant, dark-coloured, and on standing tends to separate into layers. Microscopically, we find debris of lung substance, various organisms, crystals of fatty acids, hæmatoidin, leucin and tyrosin, etc., but, on account of the presence of ferments, no elastic tissue. It must be remembered that the sputum may suggest gangrene, without that actually being present, and that the converse of this may also occur.

Abscess of the Lung.—As a rule the abscess empties itself periodically, and the patient, after bringing up a large quantity of pus, has relief for a time. An empyema or abscess perforating the lung from without gives rise to the same symptoms, and multiple dilatation of the smaller bronchi may have a similar result. The presence of elastic tissue of alveolar form may assist in the diagnosis of abscess from perforating empyema and bronchiectasis. Besides the elastic tissue organisms may be found. One of the pyogenic cocci is commonly present, or, in rarer instances, some other microbe.

In amœbic hepato-pulmonary abscess the sputum (according to Davidson) is rather mucoid than purulent, being partly watery, partly viscid and frothy; in the earlier stages red corpuscles predominate over leucocytes, giving a bright red to a dull brick-red or brownish colour. The expectoration contains numerous small, friable, cheesy masses, while microscopically various crystals, elastic fibres, and, in particular, the amœba coli can be seen.

Œdema of the Lungs.—The sputum is frothy, watery, copious, and poor in formed constituents. It is often blood-stained, but contains few red corpuscles, the coloration being due mainly to blood-pigment. Unlike most other sputa it is rich in serum albumin, and has a high specific gravity.

III. Microscopical Examination of the Sputum.—Although for the recognition of organisms staining is necessary, the examination of fresh specimens of sputum, by placing a small portion under a cover-glass, with or without the addition of a drop of acetic acid, should not be omitted. The cells may be further studied by fixing thin films for five minutes in formalin 1, alcohol 10, and staining with eosin-hæmatoxylin or eosin-methylene blue.

(1) *Cellular Elements.*—*Leucocytes* are usually abundant; they may be highly granular, or contain in their interior carbon particles or altered blood-pigment. Eosinophile cells may be found in great numbers in the sputum of asthma and of so-called eosinophilic bronchitis.

Red corpuscles retain their form for a considerable time after extrusion from the vessels, the principal change to which they are liable being loss of pigment, which passes into the ground substance of the sputum. In course of time, however, they shrink, and become otherwise altered, leaving behind pigment granules or hæmatoidin crystals. Blood corpuscles are, of course, present in all cases of hæmoptysis, but it is important to remember that a few may be found in almost every sputum.

Epithelium.—*Squamous epithelium* is derived from the mouth, pharynx, or larynx, especially about the true cords. *Ciliated epithelium* is rare; if abundant, it has probably come from the nares, since most of the ciliated epithelium of the trachea and bronchi loses its cilia when shed, and appears in the sputum in the *cubical form.*

The most important epithelial cells, however, are the so-called *alveolar cells,* although their source and significance are still doubtful. These are very common; they are round or oval cells, several times as large as a leucocyte, with one or more nuclei situated in a granular or fatty protoplasmic body. Semi-translucent, rounded myelin droplets with sharp outlines may be found in some of these cells, as well as free in the substance of the sputum. It is said that when alveolar epithelium is found in abundance it indicates a catarrhal condition of the alveoli rather than of the finer bronchi. Alveolar epithelium often contains blood-pigment, particularly in chronic venous congestion of the lung. Such "Heart cells," as they are called, are most commonly found in cases of mitral stenosis.

(2) *Lung Tissue.*—Comparatively large pieces of lung tissue are occasionally expectorated in cases of gangrene of the lung. The most important substance coming under this head is, however, yellow elastic tissue, which is pathognomonic of destructive change in some part of the respiratory tract. It may be found wherever

—as in bronchiectasis—ulceration of the bronchi is proceeding, and is sometimes present in croupous pneumonia where there is localised necrosis of the hepatised lung. It is generally (but not invariably) absent from the sputum of gangrene, owing to the development of a ferment which dissolves it. But its chief diagnostic value (though this has been somewhat discounted in modern days by the ease with which the bacillus can be detected) is in the early diagnosis of phthisis, and since elastic tissue undoubtedly sometimes precedes the bacillus in the sputum, it should be sought for in doubtful cases. The sinuous fibres are highly refractile, with well-defined outlines; they branch dichotomously, and are sharply broken off, not frayed, at the ends. As they may arise from ulceration of the bronchi as well as from changes in the parenchyma of the lung, only those which retain the alveolar arrangement justify the diagnosis of phthisis.

Where the fibres are abundant it is sufficient to place one of the denser portions of the sputum on a slide with a drop of liquor potassæ, and examine under the microscope. If they are scanty the sputum should be boiled for five minutes with an equal bulk of liquor potassæ, and diluted with several volumes of water. The deposit collected on standing, or in the centrifuge, will then contain the fibres.

(3) *Curschmann's spirals* appear as whitish, rolled-up, sago-like balls; closer inspection shows them to consist of a coiled spiral thread, which, when extended, may measure 1-2 cm. They appear to consist of cells embedded in a matrix whose delicate fibrils run spirally round a clear, bright, axial thread, which has a convoluted, zigzag course. Some spirals, however, lack the central thread. The source, nature, and mode of formation of these spirals have been much debated. They certainly arise in the bronchioles, and their spiral character is probably due to the substance of which they consist becoming twisted as it is expelled. Troup regards the axial thread as epithelial; others look upon it, with the rest of the spiral, as fibrinous. Sputum containing spirals is excessively tenacious, and not infrequently shows—sometimes in the spirals—eosinophile cells and Charcot-Leyden crystals. Spirals point to a catarrh of the bronchioles—*bronchiolitis exudativa* (Curschmann); they occur in many cases of asthma, and occasionally in bronchitis, pneumonia, and œdema of the lungs.

(4) *Fibrinous casts* may form in the respiratory passages as a result of scalds, etc., and in various infective conditions. They also occur in diphtheria, pneumonia, and plastic bronchitis. It may be impossible to make a diagnosis of their source by the microscope alone, but attention to the clinical features of the case will usually obviate difficulty in this respect. In plastic bronchitis casts of a large part of the bronchial tree may be expelled, or, in another set of cases, only short branching casts (obviously from the smaller bronchioles) are seen. The latter are associated with paroxysmal asthma-like attacks, and it is probable that although they show no spiral arrangement they are allied to true spirals. Casts consist of cells embedded in a homogeneous matrix; they may contain spirals, Charcot-Leyden crystals, or eosinophile cells. They dissolve in alkalies, and swell up on the addition of acids. The casts of diphtheria generally consist of comparatively small pieces of membrane, in which the Loeffler bacillus can be demonstrated. Casts of the smaller bronchi can often be detected in croupous pneumonia if the sputum be floated out in water; they are seldom of importance clinically.

(5) *Crystals.*—*Charcot-Leyden crystals* are colourless, sharp-pointed octahedra of uncertain chemical composition. They are insoluble in alcohol, ether, and chloroform, but dissolve in acids and alkalies. They occur (often only after the sputum has been allowed to stand for a time) in asthma and other conditions, frequently along with spirals and eosinophile cells.

Needle-like crystals of the *fatty acids*, sometimes arranged in rosettes, are common in sputa which stagnate in the bronchi. They are insoluble in acids and alkalies, but dissolve in alcohol and ether.

Hæmatoidin crystals may occur either in the cells or ground substance of the sputum. In the former case they are usually scanty, and (according to von Jaksch) point to previous rupture of a pulmonary vessel. If abundant they are suggestive of a pulmonary abscess or empyema. Among rarer crystals, *cholesterin*, *leucin* and *tyrosin* (occurring in bronchiectasis or pulmonary abscess), *oxalates*, and *triple phosphates* may be mentioned. Calcareous concretions from old tuberculous deposits may also occur.

(6) *Tumour Growth.*—In cases of intrathoracic tumour portions of the growth may, though rarely, be found in the sputum.

(7) Foreign bodies of various kinds may be expectorated.

(8) *Vegetable Parasites.*—A. *Pathogenic. Tubercle Bacillus*—Method of examination.—The suspected sputum is spread out in a flat dish—preferably black—and a thick, purulent portion is sought for. When the expectoration consists entirely of mucus, the detection of the organism becomes more difficult, but should still be attempted.

A piece of the selected portion of sputum about the size of a pin's head is then removed with a fine pair of forceps, and placed on a clean cover-glass, over which another is placed, and the two squeezed together so as to spread the sputum in a thin layer. The cover-glasses are then separated by sliding apart, and the

films are allowed to dry. They are then fixed by passing thrice rapidly through the flame of a spirit-lamp, great care being taken not to scorch them. They are now ready for staining, which is as easily accomplished by the Ziehl-Neelsen method as by any other. A few drops of carbol-fuchsin (fuchsin 1, alcohol 10, 5 per cent aqueous solution of phenol, 100) are placed in a watch-glass, and the cover-glass is floated thereupon, film side downwards. The whole is then heated until it steams, and is allowed to stand for five minutes, after which it is washed and then placed in a strong aqueous solution of methylene blue containing 20 per cent of sulphuric acid. In this it remains for 1-2 minutes, but should be removed and rinsed at intervals to see how the staining is going on. It is generally advisable to stop the process while there is yet a faint tinge of pink in some part of the film. Having finally washed the film, it is dried between layers of filter-paper and mounted in balsam.

Attention has recently been drawn to the occasional occurrence in the sputum of acid-fast bacilli other than the tubercle bacillus. The principal example is the smegma bacillus, and there are others. The following methods have been advised for distinguishing the bacillus of tubercle from these:—(1) decolourise with alcohol instead of acid; (2) decolourise with Ebner's fluid (corallin 1, absolute alcohol 100, methylene blue to saturation, glycerine 20). This is allowed to act for several minutes (Pappenheim) or preferably for four hours (Coles).

There is usually little difficulty in detecting bacilli if they are present. They appear as small, straight or slightly curved, red-stained organisms, while other bacteria, cells, etc., are bright blue. The discovery of the bacillus is chiefly of diagnostic value. On the whole, their number affords but slight aid in prognosis. Little importance can be attached to irregularities in the shapes of the bacilli: these were formerly thought to be a sign of sporulation, but this has not been definitely proved. Since the bacilli in the sputum may be few, a single negative observation is of little value, and must be repeated. In doubtful cases a little liquor sodæ should be added to the sputum, and the whole diluted after warming gently; the deposit collected on standing or in the centrifuge must then be examined.

Pneumococcus.—This may be recognised by staining films of sputum with methylene blue or carbol-thionin blue. The organisms are seen as small oval cocci about 1 μ in diameter, usually arranged in pairs or in rows of six or eight. Each pair (or diplococcus) is surrounded by a pale, unstained zone—the capsule. The diplococcus is also stained by Gram's method, but for differential staining of the capsule special methods are required. Since the pneumococcus may be absent from the sputum in pneumonia, and present apart from that disease, it is only of value as confirmatory evidence. In about 5 per cent of cases of pneumonia Friedländer's bacillus is present. It somewhat resembles the pneumococcus in form, and, like it, possesses a capsule. It is stained by the basic aniline dyes, but *not* by Gram's method. Further important staining methods are described under FLUIDS, EXAMINATION OF PATHOLOGICAL (*Bacteriology*).

Influenza Bacillus.—This is present in the sputum in influenza (in many epidemics, at least) if the respiratory tract be involved. It is a minute organism about ·5 μ long, lying singly, in groups, or in chains. It stains with carbol-fuchsin, but not by Gram's method. Its recognition is a matter rather of scientific than of practical interest.

Other organisms may be found in the sputum. *Pyogenic cocci* are present in many cases of broncho-pneumonia, and, in fact, in almost all purulent sputa. In pneumonia secondary to an infectious disease—*e.g.* typhoid, diphtheria, etc.—the specific organism may be found; more commonly, however, we have the pneumococcus. The *bacillus mallei* has been described in cases of glanders affecting the respiratory tract. Last, in cases of *pneumonic plague*, the diagnosis rests on the discovery of the bacillus.

Actinomycosis of the lung may show itself by the presence in the spit of the characteristic yellow granules composed of the club-shaped filaments of the ray fungus. The only other of the higher micro-organisms which is pathogenic in the sputum is the *Aspergillus fumigatus*, a mould to which pulmonary aspergillosis—an excessively rare disease—is due. Since other moulds are not uncommon, cultural methods are needed for the recognition of the aspergillus fumigatus. The *streptothrix* has also been found in the sputum in cases of pulmonary streptothricosis. It does not stain with carbol fuchsin, but requires the Weigert gram method for its detection. Another rare pneumo-mycosis is that due to *blastomycetes*—a yeast fungus. The organism is found in the sputum. Both pulmonary streptothricosis and blastomycosis clinically resemble tuberculosis.

B. *Non-pathogenic.*—Among these may be mentioned the putrefactive bacteria, leptothrix, and vibriones, none of which are uncommon. Sarcinæ are sometimes found in cases of ulceration of the bronchi, and yeasts and moulds may occasionally be detected. The *oidium albicans* is usually due to contamination from the mouth, though probably thrush sometimes involves the upper portion of the trachea also. Sarcinæ occur in gangrene of the lung.

(8) *Animal Parasites.*—The *Distoma pulmonale* gives rise to a form of pulmonary disease (distomiasis), endemic in Japan and Corea, and associated with hæmoptysis. The brown ova, ·1 mm. long by ·05 mm. broad, are found in the sputum. In cases of *hydatids*

involving the lung, portions of cyst-wall, scolices, or hooklets may be expectorated. Trichomonades have been found in the sputum of gangrene, and the amœba coli in the pus from an hepatic abscess. When the presence of the amœba is suspected the sputum should be examined on a warm stage as soon after expectoration as possible, when the motility of the organism will serve to distinguish it. In the resting condition it is a pale greenish, highly refractile structure, whose average diameter is from 12 to 26 μ. The body consists of a clear, homogeneous ectoplasm, and a darker, granular entoplasm. There is an eccentrically placed nucleus, for whose detection, however, staining is required. By fixing films in alcohol and staining with methylene blue the entoplasm is deeply stained, while the ectoplasm remains almost colourless. The best picture of the nucleus is given by fixing in Fleming's solution and staining with saffranin and acid fuchsin (Davidson).

Expert.—An individual who has made a special study of any subject, who has had special experience therein, and who is, therefore, qualified to give valuable evidence thereupon; as a witness, an expert may speak on matters of opinion. *See* MEDICINE, FORENSIC.

Expiration. *See* PHYSIOLOGY, RESPIRATION (*Movements in Expiration*); PULSE (*Frequency*); RESPIRATION (*Physiology, Expiration*).

Exploratory Operations. — Exploratory operations are those usually carried out, in the first place, for diagnosis, but, secondarily, for treatment, if the conditions found be such as to warrant or justify operative interference; they usually take the form of opening into a body-cavity by incision or aspiration, such as the abdomen, thorax, cranium, or spinal canal.

Explosives. *See* TRADES, DANGEROUS.

Exposure, Death from. *See* MEDICINE, FORENSIC (*Death from Cold and Exposure*).

Expressio Fœtus.—Delivery of the child by compressing and rubbing the uterus through the abdominal walls of the mother.

Expression. *See* PHYSIOGNOMY AND EXPRESSION.

Exstrophy.—The exposure of the interior of such a viscus as the bladder (vesical exstrophy or ectopia vesicæ) by defect of its anterior wall and of the anterior abdominal wall; extroversion or inversion. *See* BLADDER (*Malformations*); ECTOPIA; TERATOLOGY.

Extension. *See* FRACTURES (*Femur, Treatment*); HIP-JOINT, DISEASES (*Morbus Coxæ, Treatment*); SPINE, SURGICAL AFFECTIONS (*Spinal Caries, Treatment*).

Extensor Response. *See* BABINSKI'S SIGN.

Extern. — A patient treated outside a hospital (an out-patient), or the medical or surgical officer who is in charge of the out-patients or who resides outside the hospital.

Extirpation.—The complete removal of an organ, such as the uterus or the eye, by operative means. *See* UTERUS, NON-MALIGNANT AND MALIGNANT TUMOURS (*Treatment*).

Extracta.—Pharmaceutical preparations, liquid or solid, made by making solution of the soluble parts of the plant, etc., or by evaporating down their juices; they are classified as fresh, green, aqueous, alcoholic, ethereal, and liquid extracts; and they form a large group of official preparations. *See* PRESCRIBING; PHARMACOLOGY; *also under* the various drugs (*e.g.* Belladonna, Ergot, Gentian, Opium, etc.).

Extractives.—Matters, present in small amount and having indefinite characters, which can be separated by solvents during the analysis of organic substances. *See* PHYSIOLOGY.

Extract of Jez.—An extract, obtained from the tissues (spleen, bone-marrow, spinal cord, thymus, brain) of animals immunised to typhoid bacilli, and used in the treatment of typhoid fever.

Extra-Systole.—A heart-contraction occurring earlier than is normal; the next systole occurs after an unduly prolonged period; and the long and short period together equal the duration of two normal periods; a disturbance of cardiac rhythm or arrhythmia.

Extra-Uterine Pregnancy. *See* ECTOPIC GESTATION.

Extravasation. *See* BLADDER, INJURIES OF (*Rupture, Extravasation of Urine*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Hæmorrhage*); MENINGES OF THE CEREBRUM (*Vascular Disturbances*).

Extremity. *See* DEFORMITIES (*Upper and Lower Extremities*); FRACTURES (*Upper and Lower Extremities*).

Extroversion. *See* BLADDER (*Malformations*); ECTOPIA; EXSTROPHY; TERATOLOGY; etc.

Exudation and Exudates. *See* INFLAMMATION (*Escape of Fluid, Exudates*); EMIGRATION OF LEUCOCYTES; etc.

Eye. *See* ACCOMMODATION; ACROMEGALY (*Symptoms, Eye*); AMBLYOPIA; ASTHENOPIA; BANDAGES (*Eye Bandages*); BLINDNESS; BRAIN, PHYSIOLOGY OF (*Sensory Centres, Sight*); BRAIN, SURGERY OF (*Compression of the Brain, Eye*

Symptoms); Cataract; Colour Vision; Choroid, Diseases; Conjunctiva, Diseases of; Cornea, Diseases and Injuries; Death, Signs of (*Eye Lustre*); Diabetes Mellitus (*Symptoms and Complications, Eye Changes*); Eye, Clinical Examination of; Eyeball, Injuries of; Eyelids, Affections of; Filariasis (*Filaria loa or Dracunculus Oculi*); General Paralysis (*Symptoms, Eye*); Glaucoma; Hay Fever; Headache (*Causes, Eye*); Herpes (*Herpes Zoster, Complications, Ocular*); Iris and Ciliary Body; Lacrimal Apparatus; Lens, Crystalline; Leprosy (*Clinical Features, Eye Affections*); Meningitis, Epidemic Cerebro-Spinal (*Symptoms*); Mental Deficiency (*Mongoloid Cases*); Nose, Accessory Sinuses Inflammation (*Chronic Suppuration, Eye Symptoms*); Ocular Muscles, Affections of; Ophthalmoplegia; Orbit, Diseases of; Physiology, Senses (*Vision*); Refraction; Retina and Optic Nerve; Retinoscopy; Sclerotic, Diseases of; Strabismus; Syphilis (*Secondary, Tertiary, Eye and its Appendages*); Tabes Dorsalis (*Symptomatology, Eye Symptoms*); Teeth (*Neuralgias of Dental Origin*); Thyroid Gland, Medical (*Exophthalmic Goitre*); Vision, Field of; Vitreous Humour, Diseases of.

Eye, Clinical Examination of.

An examination of the eye may be required to discover the cause of ocular symptoms, to aid in the diagnosis of some general condition, or to ascertain the fitness of the subject of the examination for a particular occupation.

For whatever purpose the examination is undertaken it should be conducted upon a definite plan, otherwise some points may easily escape detection. It is, of course, not necessary that the whole examination should be gone through in every case. Some symptom may direct attention at once to the salient feature in the case, or an opinion may be required on some particular point.

Cases which are examined in consequence of a defect in vision usually come, in the first instance, under the notice of the ophthalmic surgeon, but in the course of his examination he not infrequently is led to the discovery of some general disease previously unsuspected. Perhaps the most frequent example of this is the diagnosis of albuminuria or glycosuria from changes in the retina. The presence of optic atrophy, or of abnormal action of the pupils, may lead to the discovery of tabes dorsalis, or disseminated sclerosis. While a diagnosis of syphilis, inherited or acquired, may be established by an examination of the cornea, iris, or choroid.

The physician not infrequently is aided in his diagnosis by an examination of the eye, as, for example, when the presence of double papillitis tends to strengthen a doubtful diagnosis of intracranial growth, or when the localisation of a cerebral lesion is aided by noting the condition of the visual fields, or abnormalities in the action of the orbital muscles or pupils.

Candidates for the Government services, for the posts of railway signalmen, or of officers on ships, and even school-board teachers, afford examples of the need for an examination of the eyes. Examinations for such purposes are often entirely neglected, or inefficiently carried out, to the great injury of the individual and the danger of the public (*vide* "Colour Vision").

Before beginning a detailed examination of the eyes a general survey of the patient should be taken, and any outstanding evidences of past or present disease or injury mentally noted. Syphilis, hereditary or acquired, is of primary importance in this respect, while among other diseases specially liable to give ocular manifestations may be mentioned Bright's disease, diabetes, and locomotor ataxy. The so-called diathesis of the patient may be important especially in relation to rheumatic and gouty conditions in adults, and to the so-called strumous affections in children. In cases of cerebral tumour the patients seldom seek advice spontaneously on account of defective vision, the examination of the eyes being usually made to aid the physician in establishing a diagnosis.

The gait and bearing of the patient should always be noted. Unsteadiness of gait may be an indication of some severe nervous lesion, but may also be due to the vertigo produced by recent paralysis of an ocular muscle. In the latter case the head is often held obliquely, being turned in the direction of action of the paralysed muscle. A patient with nuclear cataract will often walk towards the window with his head slightly bent, and his hand shading his eyes in order to get the pupils to dilate so as to enable him to see past the central opacity. Patients with double ptosis often walk with the head thrown backwards and the brows wrinkled, so that they may see from under the overhanging lids.

The eyes should next be looked at from a distance of a few feet in a good light, and any evident abnormal appearances noted. The posi-

tion of the eyelids, the size and shape of the palpebral fissures, and any undue retraction (enophthalmos) or prominence (exophthalmos) of one or both eyes should be specially noticed. Abnormal movements of the eyes, such as nystagmus, or misdirection of one of the visual axes (strabismus), will, if marked, be evident at a glance.

An inquiry into the symptoms complained of by the patient should now be made, and will often greatly aid the surgeon in diagnosis and treatment.

1. *Defects in Vision.*—The acuity of vision should be measured and recorded by the method given below. At the same time the range of accommodation and convergence should be noted. It should be ascertained if the prolonged use of the eyes upon near objects caused dimness, pain, or discomfort (asthenopia). In the case of any real lowering of the vision an attempt should be made to ascertain whether the defect is of recent origin, if its onset was sudden or gradual, and if at the time of the examination it is progressive or stationary. It must be borne in mind in this connection that a patient often suddenly, and by accident, discovers that one eye is defective, and usually dates the onset of the defect from that moment.

2. *Diplopia.*—Although this symptom is often referred by the patient to one eye it is always binocular, and due to deviation of one of the visual axes (squint) caused by partial or complete paralysis of an ocular muscle. In slight cases in which the deviation is not obvious the diplopia becomes a most valuable means of localising the lesion (*see* "Ocular Muscles"). Concomitant (non-paralytic) squint hardly ever leads to a complaint of diplopia. True monocular diplopia is so rare that it need not be considered.

3. *Distortion of Objects* (*Metamorphopsia*).—This arises chiefly under three sets of conditions:—(1) Irregular astigmatism; (2) Exudation into the retina and choroid, especially in the macular region, interfering with the regular arrangement of the percipient elements; (3) Anomalies of accommodation causing errors of judgment as to the distance and size of objects.

4. *Subjective Sensations of Light; Spots before the Eyes; Anomalies of Colour Vision.*—A constant sensation of dazzling is sometimes associated with inflammatory lesions of the optic nerve or retina due to morbid stimulation of the conducting or percipient apparatus. Similarly in the early stages of retinal detachment sudden flashes of light are often complained of. Sensations of light (phosphenes) are readily produced by pressure on the eyeball behind the ora serrata, and are due to the same cause. They are referred to the part of the visual field corresponding to the area of retina stimulated; thus pressure below will give a phosphene above, and so on. The peculiar subjective visual phenomena accompanying migraine depend probably on stimulation of the higher centres connected with vision. In opacities of the media the appearance of a dark object or objects may be complained of. If the object is described as moving or floating the opacity is in all likelihood situated in the vitreous. Real opacities visible with the ophthalmoscope must not be confused with the floating specks (*muscæ volitantes*) so often noticed by patients, especially if an evenly illuminated surface as the sky be looked at. These have no pathological significance. Blind areas on the visual field occur in many forms of eye diseases, constituting scotomata. If the blind area is noticed by the patient the scotoma is said to be positive, and is caused by a lesion situated in front of the percipient layer of the retina. A negative scotoma can only be discovered by the perimeter, and is due to loss of function in the percipient layer. The presence of rings of colour round lights is a well-known premonitory symptom of glaucoma.

The occupation and habits of the patient have often an important relation to symptoms complained of or to actual diseases observed. Thus miners are liable to nystagmus, which has been attributed to strain of the ocular muscles produced by their working in cramped attitudes and also to the defective illumination. Asthenopia is much more frequently complained of by those who have to use the eyes for near work, as in reading, writing, sewing, etc. Poor and dirty surroundings predispose to all affections of the lids and conjunctivæ, abuse of tobacco and alcohol frequently gives rise to impaired vision, the so-called toxic amblyopia, and workers exposed to the action of lead, carbon-bisulphide, and other substances may present ocular symptoms as a manifestation of the poisoning of the system.

A detailed Examination of each Eye should now be made.—The appendages of the eye—the lids and lachrymal apparatus—should first be examined. Secretion forming crusts on the edges of the eyelids indicates blepharitis, the only condition that can be mistaken for it being pediculi (ptheiriasis palpebrarum). Examination with the pocket lens would at once show the nits adhering to the lashes. The shape and position of the eyelids should then be noted. The margins may be everted, "ectropion,"—or inverted, "entropion." The former may result from old blepharitis, chronic conjunctivitis in old people, or it may result from the traction of cicatrices on the face. Paralysis of the facial nerve causes a slight ectropion of the lower lid, and lachrymation owing to the punctum falling away from the eye.

Without any entropion being present a few lashes may be displaced so as to turn in and brush against the cornea. As such hairs are often very fine they are easily overlooked, unless a lens is used to examine the lid margin. Occa-

sionally a source of conjunctival irritation is an eyelash impacted in one canaliculus, the free end coming into contact with the cornea.

The condition of the lachrymal apparatus should next be ascertained. The lachrymal gland cannot usually be felt unless it is pathologically enlarged. Its edge can then be made out as a smooth, hard body immediately below the outer third of the upper margin of the orbit. Obstruction in the drainage apparatus is, on the other hand, excessively common. The punctum may be congenitally absent, it may be occluded by cicatrisation, or displaced from ectropion. In all these conditions the tears, being unable to escape by the natural channels, run down the face.

More frequent and more serious than these conditions is obstruction of the nasal duct. When this is present the tears accumulate in the lachrymal sac, and regurgitate into the conjunctival cul-de-sac when pressure is made over the lachrymal sac in a direction backwards, upwards, and inwards. Sometimes the obstruction is incomplete, and the contents of the sac can be pressed down the duct. The contents of the sac may consist of tears only, of tears mixed with mucous secretion (hence the term "mucocele"), or of pus.

The existence of a mucocele is important in view of any operative proceeding on the eye, as the wound may easily become infected with pyogenic organisms.

Epiphora, or watery eye, is common to all affections of the lachrymal drainage system. It may, however, be present merely as the result of over-secretion of tears. This may be due to an error of refraction, but it is not infrequently met with without any ascertainable cause. The conjunctiva in these cases appears to be hypersensitive, and on its exposure to cold air a reflex secretion is brought about (*see* "Lachrymal Gland").

We now return to the eyelids to consider the condition of their mucous surface. To expose this it is necessary to evert the lids. In the lower lid this is effected by merely drawing the free edge downwards, but to evert the upper lid neatly and painlessly requires some little dexterity.

Either of the following two methods may be employed—(1) The surgeon stands behind the patient, the latter pressing his head against the surgeon's chest. The patient should look down, while the surgeon holding some round body, such as a probe or pencil case, lays it gently horizontally on the lid, just above the upper edge of the tarsus. With the thumb and forefinger of the other hand he takes hold of the free edge of the lid and draws it downwards and forwards. The probe being held firmly, the lower edge of the lid can now be drawn up and the lid everted.

(2) The surgeon faces the patient, and using his right hand for the patient's left eye, and *vice versa*, he places the pulp of his index finger on the lid, and uses this as the fulcrum, while the thumb is placed under the free edge of the lid, which it pushes forwards. This method requires more practice than the other, but is somewhat quicker, and it can be applied to both eyes at once. With either it is important that the patient should look down in order to relax the levator muscle.

The conjunctival surface of the lid thus exposed should be moist and perfectly smooth, except over the prominences caused by the ends of the tarsus, where a few minute papillæ can generally be seen. The colour is pale red from fine blood-vessels. As regards shape, it is concave from side to side, but from above downwards it should be nearly flat. Parallel with the free edge, and two millimetres from it, is a fine groove, which is a frequent lodging-place for foreign bodies. The cul-de-sac should also be explored. To expose the upper the patient should look well down while the lid is kept everted. If the surgeon will then press the globe gently backwards the upper cul-de-sac will usually roll forward. The tissue of the cul-de-sac is loose and contains lymphatic bodies which, when swollen, look like grains of boiled sago. These "follicular granulations" are sometimes evidence of malnutrition, but they are occasionally met with in persons who appear to be in perfect health. It is possible that their presence increases the liability to any contagious conditions, but they are not of themselves of any other importance.

The palpebral conjunctiva may be velvety from chronic inflammation, it may be covered by sticky mucus or with pus. In catarrhal or purulent ophthalmia the secretion may be coagulated into shreds or membranes; but this condition must not be confounded with the much rarer, but more serious, diphtheritic inflammation. In the latter a raw surface is left on the removal of the membrane, and bacteriological examination will show the specific organisms.

It is probable that all abnormal conjunctival secretions are contagious to some degree, but their virulence varies greatly. Next to the diphtheritic cases the most actively contagious are those in which the gonococcus is found in the secretion.

Bacteriological examination is of great diagnostic value in some cases. It must, however, be borne in mind that the conjunctiva being constantly exposed to the air affords a lodgment for many micro-organisms, which may have no causal relations to affections of the conjunctiva since they are present under normal conditions.

The lid linings may be affected with trachoma—granular lids; in such cases the lid is rough from "granulations," which vary in appearance, some being round or oval greyish bodies like the follicular bodies already described, others

large truncated elevations of the colour of the normal conjunctiva. At a later stage these become firmer and less vascular; finally they may disappear, leaving depressions, so that the lid has a honeycombed appearance. At this stage cicatricial contraction takes place, the groove near the free margin becomes deeper, and the whole lid becomes more curved from above downwards, so that the lashes may brush against the cornea—entropion.

In all cases of trachoma the cornea should be carefully examined, since vessels are very apt to encroach on its upper margin—pannus.

In cases of trachoma of old standing the roughness may disappear, but there are bands of cicatricial tissue beneath the conjunctiva, and by the contraction of these the lid is shortened and curved, the palpebral fissure narrowed, and the depth of the culs-de-sac diminished. At the same time the surface of the conjunctiva is unnaturally dry, owing to the destruction of its glands.

A somewhat similar condition is occasionally seen independently of trachoma. This has been called "essential shrinking of the conjunctiva," or more recently "pemphigus," because in many cases the history has pointed to a connection between this disease and pemphigus of other mucous membranes. The disease is progressive, and eventually the whole of the culs-de-sac become destroyed, the lids being in contact with and adherent to the globe, and the cornea dry and opaque.

The ocular conjunctiva next claims our attention. This should be quite transparent, so that the sclerotic is visible through it; it should contain only a few visible vessels, and should be freely movable over the sclerotic. There are a considerable number of small vessels which readily become distended and visible on exposure to any irritant hyperæmia of the conjunctiva. The distinction between this and simple conjunctivitis is one merely of degree.

It is a matter of considerable importance to distinguish between conjunctival and ciliary injection. The former is due to distension of superficial vessels which supply the conjunctiva only, the latter to distension of fine vessels round the cornea, which are non-perforating branches of vessels which supply the iris and ciliary body.

When the vascularity is purely conjunctival the vessels are distributed over the whole surface; they can be emptied by slight pressure with the finger through the eyelid, and they can be moved with the conjunctiva over the surface of the sclerotic.

In ciliary injection the affected area is limited to a zone about a quarter of an inch wide round the cornea. The vessels can only be emptied by firm pressure, and they are immovable.

The distinction in typical cases is therefore easy, but long-continued conjunctival injection may cause some distension of the deeper vessels with which they communicate, and ciliary injection is usually complicated with conjunctival.

If the injection is purely conjunctival inflammation of the iris may be excluded. On the other hand, the presence of ciliary injection should always raise a suspicion of inflammation of iris, cornea, or ciliary body.

In catarrhal conjunctivitis, or catarrhal ophthalmia, the palpebral as well as the ocular portions are affected. In addition to the vascularity there is a secretion of mucus, which may merely be sufficient to render the surface sticky, or may be more abundant. Since the corneal epithelium is continuous with that of the conjunctiva it is often involved, superficial ulcers being formed.

Phlyctenular conjunctivitis, or strumous ophthalmia, is common in children; it may be associated with the catarrhal form, or may occur independently of it. Small round white elevations, about the size of millet seeds, appear usually at the margin of the cornea—"phlyctenulæ." They may lie at some distance from the cornea, or there may be on the cornea itself small round superficial ulcers which are of the same nature.

Photophobia and Blepharospasm.—In any inflammatory condition of the eye the photophobia may be so intense that it leads to a reflex contraction of the orbicularis, which may render examination of the eye difficult or impossible. Photophobia occurs more readily in children than in adults, and is often present in them in a severe form when careful examination of the eye fails to detect more than a slight catarrhal ophthalmia. As a rule, however, severe photophobia indicates that there is some ulceration of the cornea, or that this has been present till recently. The dread of light, however, sometimes persists long after the cause has disappeared, and seems in such cases to be due to the fear that light will hurt, as it used to do, rather than to any actual pain. When the photophobia is of this mental character it can often be overcome by placing the child with its face away from the light, and then reflecting light on to the eye from a mirror.

When the spasm of the orbicularis has lasted long a mucous chink forms at the outer canthus, and relief may sometimes be obtained by dividing the fibres of the orbicularis through this.

When it is desired to examine the cornea in infants the child should be laid on its back on the nurse's knee, with its head towards the surgeon, who is also seated, so that he can steady the child's head between his knees. In cases of purulent or diphtheritic ophthalmia it is well for the surgeon to protect his own eyes by goggles. The lids can now be separated by placing a thumb upon each near the margin and gently drawing them apart. When the lids are swollen care must be taken, by keeping them

pressed against the cornea, that they do not become everted. It may, however, be impossible to see the cornea in this way owing to its being rolled upwards. In such cases a general anæsthetic should be employed.

In cases of purulent ophthalmia examination of the cornea is rendered difficult by the swelling of the lids. In infants the swelling, although great, is soft, and does not, as a rule, offer any serious difficulty, but in adults it may be so hard and brawny that it is impossible to separate the lids till it has been reduced.

Examination of the Cornea.—The chief methods available are—(*a*) Direct inspection; (*b*) Focal (*i.e.* concentrated) illumination; (*c*) Observation of images reflected from its surface.

(*a*) Direct inspection will often render further examination unnecessary. If the cornea be bright, its surface smooth, if there be no haziness in any part, and its shape be normal, it is free from any pathological conditions. This examination, however, will not exclude abnormalities in its curvature sufficient to interfere with vision by producing astigmatism. (*See* "Refraction.")

The average transverse diameter of the cornea is 11·6 mm. (Priestley Smith). The vertical meridian appears smaller because the scleral opacity encroaches on it. An abnormally small cornea probably indicates a predisposition to glaucoma. Very large corneæ are only met with in association with enlargement of the whole eyeball (buphthalmos); in such cases the anterior chamber is increased in depth, and the sclerotic round the cornea has a bluish colour.

This same discoloration in adults is evidence of previous attacks of cyclitis, or inflammation of the ciliary body.

An alteration of form, conspicuous in pronounced cases, but easily overlooked in the slighter degrees, is that known as "kerato-conus" or "conical cornea." In this condition the whole cornea becomes unduly prominent, but the central part is altered more than the periphery, so that the curve is no longer approximately spherical, but may be described as forming a cone with a rounded summit. Viewed from the front, the sharp curvature of the central portion renders it bright and conspicuous as if there were a drop of water lying on it. Viewed from the side the conical shape is quite evident. In cases of long standing the central part may be cloudy.

The slighter degrees can only be recognised by the irregularity of the shadow in the shadow-test, and by the distortion of reflected images,—Placido's disc showing this well.

Impaired transparency of the cornea may be due to the scarring of former ulceration, to keratitis, or to infiltration connected with an existing ulcer. It is therefore a matter of importance to ascertain whether there is any breach of surface. Usually there is no difficulty in deciding this by focal illumination, but in cases of transparent ulcer, or recent abrasion, the lesion may be overlooked. In such cases the recent introduction of fluorescine has proved of great service. A small drop of this placed on the cornea in a few seconds stains it an emerald-green wherever the epithelium is absent. By its use it is also very easy to note alterations in the size or shape of an ulcer, and to tell when it has become covered by epithelium.

(*b*) *Focal Illumination.*—The patient is seated in a darkened room, the light being placed about 20 inches to the side and slightly in front of the patient's head. The observer, by the aid of a lens of $2\frac{1}{2}$-3 inches focus, held perpendicularly in the path of the rays and at its focal length from the eye, concentrates a cone of light on the cornea. By slight movements of the lens or patient's eye, the different areas of the cornea are illuminated as desired, the surrounding parts remaining in deep shadow. Sometimes it is advantageous to employ a second lens as a magnifying-glass to examine still more in detail the illuminated area. This method is of the very greatest value in the minute examination of corneal lesions, *e.g.* ulcers, and in demonstrating nebulæ, foreign bodies, etc. It is also largely employed for the examination of the whole anterior segment of the eye, including the anterior chamber, iris, lens, and may even be utilised for the most anterior part of the vitreous.

(*c*) By examining carefully the image of some external object of regular outline, *e.g.* the bars of a window frame, as seen reflected from the patient's cornea, we can see whether there is any distortion of outline produced, indicating some irregularity of corneal curvature. Placido's disc consists of a series of concentric black rings on a white card which is perforated at the centre. The observer, looking through this aperture at the image of the disc reflected from the patient's cornea, will observe any irregularities of curvature in the concentric circles. In irregular astigmatism their contour will be irregularly altered, while in regular astigmatism the circles will assume an elliptical outline, the long axis corresponding to the meridian of least curvature.

For the exact measurement of the corneal curvature, and especially for the determination of corneal astigmatism, the ophthalmometer is used. (*See* "Refraction.")

The sensitiveness of the cornea may be impaired. This may be ascertained by touching the surface gently with a thread.

Anterior Chamber, Iris, and Pupil.—The depth of the anterior chamber will be best appreciated by the aid of focal illumination, the light being concentrated on the corneal margin. Swelling or advancement of the lens will make the anterior chamber shallow; the former condition is well exemplified by some cases of cataract before they are ripe, and the latter by

glaucoma. The anterior chamber is of course absent when the aqueous is evacuated by injury or operation, and the iris is then seen to lie in contact with the cornea till the aqueous accumulates. When the lens is absent or dislocated backwards, the iris, deprived of its support, will recede from the cornea, and the anterior chamber will appear deepened. At the same time the iris will generally be seen to quiver when any quick movement of the eye is made (irido-donesis). Occasionally, in severe blows or injuries of the eyeball, the iris is partly torn from its attachment (irido-dialysis).

In iritis there is often considerable turbidity of the aqueous, and in many cases of septic corneal ulcer a fibrino-purulent exudation accumulates at the lower part of the anterior chamber (hypopyon). Occasionally blood is present in a similar situation (hyphæma).

The surface of the iris should be bright, and the details of its structure clearly visible. Turbidity of the aqueous, of effusion of lymph into the substance of the iris, renders its surface dull, and blurs the details.

The actual size of the pupil varies in different individuals, it usually becomes small in old age. An extremely small pupil—"myosis"—is often an indication of locomotor ataxy.

The pupil should be circular and its outline regular. Increased intraocular tension often causes the pupil to assume an oval form with the long diameter transverse. Irregularities of outline point to the presence of adhesions between the iris and the lens capsule—"posterior synechiæ." These will be rendered more evident by the use of a mydriatic.

The pupil should dilate slowly on being shaded from the light, and on exposure to light it should contract quickly, and oscillate slightly before coming to rest. This is called the "direct action."

The pupils should also contract in association with convergence of the visual axes—"associated action"; and the centres being connected, the two pupils should act together—"consensual action."

The reaction to light may be lost while the associated action is present. This is known as the "Argyll-Robertson pupil." It is usually but not always associated with myosis, and is an indication of locomotor ataxy.

Contraction of the pupil is governed by the third nerve, dilatation by the sympathetic. Paralysis or irritation of either nerve will therefore produce corresponding variations in its size or mobility. Its action is also mechanically interfered with by posterior synechiæ. The sympathetic nerve also supplies the involuntary muscular fibres that widen the palbebral fissure, hence differences in the size of the pupil produced by the affections of the sympathetic are accompanied by corresponding alterations of the palpebral fissure.

For the light-reflex to be present it is necessary that the reflex chain should be complete. This consists of the optic nerve which conveys the stimulus to the nerve-centre, the centre itself, and the fibres of the third nerve which go to the iris. Consciousness forms no essential factor in the act, so that it is possible for vision to be absent while the reflex action persists. This is sometimes seen in uræmic poisoning.

In cases of hemianopsia, if the pupils react when light falls upon the blind part of the retina—"Wernicke's sign"—the lesion is on the proximal side of the primary optic ganglia.

Crystalline Lens.—Opacities in the lens, and, under favourable conditions, those in the anterior part of the vitreous, can be seen by focal illumination.

Opacities in the lens viewed in this way appear white. Opacities on the anterior capsule, which are caused by deposits of lymph from former iritis, may be rust-coloured.

Any opacity on lens or capsule, when viewed with the ophthalmoscope, appears black against the bright red background of the fundus-reflex.

When there is advanced cataract it is sometimes of importance to ascertain if the cataract is "ripe," *i.e.* if the opacity comes right up to the level of the iris. When there is a layer of transparent lens between the opacity and the iris, light thrown obliquely into the pupil does not illuminate the whole area, but the iris throws a crescentic shadow on the lens on the side from which the light comes.

The reflections from the posterior and anterior surfaces of the lens are sometimes used as tests to determine its presence or absence. A candle is held at arm's length in front of the patient, and at about an angle of 30° with the middle line. The observer looking into the eye from the side opposite to the light, and at about the same angle, sees the corneal and the two lenticular reflexes. The one from the anterior surface is dim and large, and moves in the same direction as the light, while that from the posterior surface is smaller, bright, and moves in the opposite direction to the light. The presence of these images proves, of course, the presence of the lens.

The depth of any opacity in the media, anterior to the posterior pole of the lens, may be readily judged by the displacement it appears to undergo relatively to the pupillary margin when it is viewed by the observer, first directly from the front, and then from the side. An object situated in front of the plane of the pupil, *e.g.* on the cornea, will appear to move towards the pupillary margin in a direction opposite to that in which the observer moves his head; an object situated in the plane at the pupil will not alter its position, while an object lying behind the pupil will appear to approach the margin of the pupil moving in the same direction as the observer's head. The amount

of displacement which the opacity undergoes gives an indication of its distance from the plane of the pupil.

For examination of the deeper parts of the eye the ophthalmoscope is required. (*See* Retinoscopy.)

Estimation of Intra-ocular Tension.—The only principle on which it seems possible to measure the intra-ocular tension in the living eye is that of gauging the resistance which the globe offers to external pressure. It is evident that any method based on this principle cannot accurately fulfil its purpose, since the total resistance is due not only to the intra-ocular tension, but also to the resistance offered by the tunics of the eye, which will depend upon their thickness.

There are two ways in which the resistance of the eye to pressure may be expressed numerically. A known pressure may be employed, and the depth of the depression made in the surface of the globe be measured, or the pressure required to produce a depression of constant depth may be measured. Mechanical appliances, called "manometers," have been constructed on both principles. The best is that invented by Mr. Priestley Smith, in which a constant pressure of 15 grammes is employed, and the depression produced is indicated in tenths of a millimetre. Most surgeons, however, produce pressure with the fingers. In practised hands this probably gives as accurate a result as any other method, while the novice would be equally likely to be misled by any mechanical contrivance.

To estimate the tension with the fingers the surgeon should face the patient. The latter should then look down, while the surgeon places his two index fingers on the upper lid, a little above the position of the cornea. With one of the fingers pressure is alternately made and relaxed, while the other, kept in position, steadies the globe. After some practice on normal eyes it is possible to become familiar with the sensation produced by treating the eye in this way, and to appreciate slight degrees of hardness or softness. It is obvious that the method requires experience, that it allows wide scope for individual differences, and that it affords no real basis for expressing the result numerically. An attempt was, however, long ago made by von Graefe in this direction, and his method is still followed. Normal tension is expressed as Tn. An eye which is perceptibly harder than normal is said to have $T+1$. If there is stony hardness, preventing any sensation of dimpling, T is $+3$, while $T+2$ is midway between 1 and 2. In the same way different degrees of softness are indicated by the signs $T-1$, -2, or -3.

The estimation of increased tension is mainly useful as an indication of glaucoma. Any considerable excess, if long continued, inevitably leads to serious consequences. Even so slight an increase as to be overlooked even by experienced observers will, if it persist for some months, produce glaucomatous cupping of the optic disc, while a great increase will cause marked symptoms immediately, and may produce complete blindness in a few hours. This is due to the loss of function in the retina from pressure upon its delicate structure, and to the impediment to the circulation in the choroidal and retinal vessels, arresting the normal process of secretion and removal of fluid. As a consequence there is violent pain radiating along the branches of the first division of the fifth nerve, intense injection of the superficial vessels of the eye, a fixed and dilated pupil, and great turbidity of the intra-ocular fluids, preventing ophthalmoscopic examination.

Between these extremes there are various degrees of increased tension causing subacute glaucoma.

Diminished intra-ocular tension, unless due to a temporary cause, such as the escape of fluid through a wound, is no less serious, since it indicates that the secreting power of the ciliary body, which is the source of supply of the intra-ocular fluid, is impaired. It predisposes to detachment of the retina, and is likely to be followed by shrinking of the eye, which then probably becomes tender, injected, and painful, and a possible source of danger to the other eye.

Visual Acuity, indicated as "V," is expressed by a fraction showing the relation between the vision of the patient and that of an arbitrary standard which is taken as the normal. The standard has been arrived at by an examination of a considerable number of normal eyes, and represents the average vision of a healthy adult eye. In children the vision is somewhat above the standard, and in old age below it, while there are also individual variations depending partly on practice, and partly on other conditions which are unknown to us.

The standard depends on the size that the retinal image of an object must have in order that the form of the object may be recognised. The size of the retinal image depends upon the size of the object and its distance from the eye. If lines be drawn from each extremity of the object through the optical centre of the eye to the retina, it will be seen that objects subtending the same angle have the same sized retinal image. The angle made by such lines at the optical centre is called the visual angle. If letters be taken the thickness of whose component lines is not less than one-fifth of the diameter of the whole letter, it will be found that an eye can easily recognise the form of the letter when it subtends a visual angle of five minutes ($\frac{1}{12}$th of a degree).

The test types known as Snellen's, which are those generally used, consist of a series of letters

of different sizes, and to each size is given a number indicating the distance at which it subtends an angle of 5 minutes. The distance must be sufficient to exclude the use of the accommodation by the normal eye. Usually 6 metres is chosen, and the lowest line is arranged for this distance and numbered 6. As, however, a distance of 6 M cannot always be obtained indoors, some sets of test-types are arranged for 5 M, the smallest letters being numbered 5. To test the patient's vision it is only necessary to ascertain which are the smallest letters that he can see at a known distance. The result is then recorded as a fraction, the numerator being the number of metres from the test-types, and the denominator the distance at which the type read should be visible to a normal eye. For example, at 6 M No. 9 is read $V = \frac{6}{9}$ths. At 6 M No. 6 is read $V = \frac{6}{6}$ or normal. If nothing can be read at 6 M the patient may be taken nearer till the top letter No. 60 is read. If this is a 3 metres $V = \frac{3}{60}$. In each case the fraction might be reduced, but if left as it is it indicates not only the relation of the patient's vision to the normal standard, but also the conditions under which the test was performed.

Although the use of Snellen's types forms in all cases a convenient method of noting the patient's vision, it does not give the acuity of vision unless any existing error of refraction is first corrected. This is the case to some extent in all forms of ametropia, but it is obvious that in myopia, the test-types being beyond the range of the patient's vision, he cannot be expected to see them, and the inability to do so does not indicate a lowered acuteness of vision. In such cases the types may be brought within the patient's far-point by means of a concave lens, or the types themselves may be brought near, but in the latter case it will, of course, be necessary to have smaller letters, so that they may subtend an angle of 5′ at the required distance. Snellen's leading types are arranged with this object, and are numbered from 4·5 to 0·3, the figures in each case indicating the distance in metres at which the type subtends at an angle of 5′. The vision by the use of these types could be noted in the same manner as the distant vision, the numerator of the fraction being the distance at which the type is held, and the denominator the distance at which the type read should be legible. These reading types, however, give less accurate result than those employed for distance, because it is difficult to get the letters of the proper size and their component parts properly related, and also because in a short distance a slight inaccuracy of measurement has more effect than when the distance is greater.

Jaeger's reading test-types are more commonly used for near vision, but these are numbered without any reference to the distance at which they should be visible.

If the patient cannot, after correction of any refractive error, read the largest of the distance types, his vision may be recorded by bringing him nearer until he can recognise the largest type. If he sees the letter marked 60 at 3 metres his vision is $\frac{3}{60}$. In cases where the vision is still further reduced the patient should be asked to count fingers in a good light, and the distance at which this can be done correctly gives a record of his visual acuity, *e.g.* RV = fingers at 2 metres. If fingers cannot be counted the hand should be moved from side by side in front of the patient's eye at a distance of a foot or so, and the patient asked to indicate the direction of movement. V = hand-movements. Finally, by shading or exposing the eye the ability of the patient to distinguish between gradations of light and shade may be ascertained, or the patient seated in a darkened room may be asked to distinguish when a light reflected from the ophthalmoscopic mirror strikes the eye, and further to state the direction from which the light comes. (Perception and projection of light.)

In testing illiterates and young children types have been devised corresponding in size to Snellen's, but all consisting of the letter E turned in various directions—up, down, to right or left. The letters are pointed out to the patient, who is asked to indicate in which direction the letter lies. Another method on a different principle is the dot-counting test in use in the army. With very small children it is often useful to show them small objects, as coins and sweets, and note whether the child recognises and clutches at the object. Young babies will follow with their eyes a light flashed on them from a mirror, and this also is a useful method in determining whether a baby is blind or not.

After recording the visual acuity as found by Snellen's types, it is often necessary to determine the nearest point of distinct vision in one or both eyes. This is done by measuring the shortest distance from the eyes at which a small, well-defined object, as small type or fine threads, can still be seen distinctly without blurring of their outlines. This point is called the near point. In moderate degrees of myopia the far point can be similarly localised by measuring the greatest distance at which a small object can still be seen distinctly. (*See* "Refraction" and "Accommodation.")

The power of convergence may be estimated by causing the patient to fix a small object held in the middle line and gradually brought nearer the patient until one or other eye is seen to give up the convergent movement. The convergence is defective if the patient cannot easily converge on a point 10 cm. distant from the root of the nose in the middle line. For more exact estimation Landolt uses a fine line of light, and the patient is asked to state the

moment at which diplopia appears as this fixation object approaches the eyes. The appearance of diplopia indicates that one eye has ceased to fix the object.

The examinee may in some cases have a reason for making out that the vision of one eye is worse than it really is, or he may even feign monocular blindness. In all cases in which any malingering is suspected the surgeon should pretend to direct his attention solely to the eye which is not complained of. By placing in front of this some glass with which it is impossible for the patient to see, and making him read the types, he can generally ascertain exactly how much sight there is in the eye which is alleged to be defective. In such cases also a prism, placed before the admittedly sound eye with its base upwards or downwards, will produce diplopia.

Another test, having the same object, consists in looking at a word composed of red and green letters arranged alternately, through similar red and green glasses placed one before each eye. The colours are complementary: the green can only be seen through the green glass, and the red through the red. If all are seen, therefore, both eyes are being used.

This test is also useful after operations for squint, to see whether the patient has regained binocular vision.

A much better test is afforded by the "glass-rod" of Maddox. A small cylinder of glass is fixed behind a narrow slit which is parallel to it. This is placed horizontally in front of one eye, and a candle placed at 6 metres is viewed through it. The light is seen as a vertical streak. The other eye sees the light in the natural way. The images are so dissimilar that no effort is made to combine them, the visual axes therefore assume the position of rest. By placing a graduated screen behind the candle the position of the line as compared with the flame can be made to give the angular deviation of the visual axis.

For convergence in near vision Graefe's well-known line and dot test may be used. This will be described under "Ocular Muscles."

A sudden and complete failure of vision occasionally occurs in one eye, without any objective signs, from acute retrobulbar neuritis. As this occurs most frequently in young women, and is often followed by complete recovery, it is sometimes mistaken for hysteria. While the blindness persists the complete absence of direct action of the pupil should prevent such an error from being made.

Colour-Vision.—Careful investigation of the state of colour-vision is frequently of the greatest importance from the point of view of diagnosis. A familiar example is seen in toxic amblyopia from tobacco (*vide* "Amblyopia"). Apart from its use as a diagnostic measure, great importance is attached to it in connection with various occupations, *e.g.* railwaymen, seamen. This subject is elsewhere fully discussed (*vide* "Colour-Vision").

Examination of the visual fields often affords important indications, and should never be omitted in cases of failure of vision, of which the cause is obscure, or when disease of the nervous system is suspected. The examination should discover the peripheral limitation of the fields, and the existence of any abnormal gaps in it (scotomata) either for white or colour.

To be complete, it should also include the fields for the primary colours, but in the majority of cases this is not required.

Any abnormality of the visual fields can be detected without any special apparatus, in the manner that will be described presently, but to record the result accurately a *perimeter* is necessary.

Various modifications of the perimeter have been designed, but its essential features are a quadrant, graduated in degrees up to 90°, which can be revolved round the point marked zero; and some arrangement by which the eye under examination can be kept in the centre of the circle of which the graduated bar forms a quadrant, so that its visual axis is directed to zero. If in these circumstances the quadrant be revolved, it evidently will describe a hemisphere in the centre of which is the eye under examination. For recording the results a chart is required. This consists of a series of radii indicating the various positions of the quadrant, and of concentric circles indicating angular degrees on each radius. The field is recorded by marking on a sufficient number of the radii the point at which the test-object becomes visible, and subsequently connecting these points by lines.

During the examination care must be taken that the eye under examination gazes steadily at zero, commonly called the "fixation point," where there should be fixed some small but conspicuous object. The test-object should always be moved from the blind towards the seeing area. If it is removed in the reverse direction the retention of the mental image will make the field appear larger than it really is. The test-object should not be moved too rapidly, and care should be taken that its movement is not audible.

Various devices are in use for facilitating the recording of the results, some perimeters being called "self-registering," because in them the record is made by some simple movement, as by pressing the chart against a pricker.

Carefully prepared perimeter charts often afford a most valuable means of seeing at a glance the progress of a case.

For the purpose of differential diagnosis, however, the following simple plan, which requires no special apparatus, will suffice.

The surgeon faces the patient at a distance

of about three feet. The patient is directed to look with the eye under examination fixedly at the eye of the surgeon which is opposite to it (*i.e.* the patient's right eye at the surgeon's left, and *vice versa*). The other eye both of patient and surgeon should be closed. It is now easy for the surgeon to detect any wandering of the patient's eye. The test-object may be a square of white paper, mounted on a black handle, or better, a long pin with a white porcelain head, such as is used for fastening on bonnets. This forms a conspicuous white object, is always clean, and can easily be obtained in various sizes. The surgeon holds the test-object midway between himself and the patient, but so far out of the line of vision that it cannot be seen; it is then gradually approached towards the line of vision, and the patient is told to give a sign the moment that he becomes aware of its presence. The surgeon's own field affords a very useful check and standard of comparison. This proceeding is repeated in different meridians, and a defect in any direction can be readily noted.

An examination for central colour scotoma should never be omitted in obscure cases. For this purpose a small red or green paper is generally used (red sealing-wax is excellent for the red). To a normal eye the colour should become more vivid as the central region is reached; if a colour scotoma is present the colour will disappear or become dull. Care must, of course, be taken that the normal blind-spot, corresponding to the optic nerve entrance, is not mistaken for a scotoma.

The limit of the field in normal vision varies slightly in different individuals. Roughly speaking, it may be said to extend externally nearly to 90°; downwards to 65°; the upper and inner limits vary with the amount of elevation of the upper lid, and the prominence of the nose. An average measurement is 55° for each.

Indications afforded by abnormal visual fields.—The following may be cited as instances in which assistance is given by perimetry, but the list is not intended to be complete.

Concentric contraction may indicate atrophy of the optic nerve or retina (the latter including "retinitis pigmentosa" and allied conditions).

Contraction in all meridians, but most on the inner side, occurs in chronic glaucoma. Loss of the upper field is common in detachment of the retina.

Loss of the temporal half of each field, "bitemporal hemianopsia," indicates a lesion at the optic chiasma. Loss of the corresponding half of each field (*i.e.* both right or both left halves), right or left hemianopsia, indicates a cerebral lesion on the side opposite to the blind areas.

Lateral hemianopsia is nearly always referred by the patient to the eye towards the blind side.

Night-blindness, or excessive lowering of the visual acuity when the light is diminished, is met with in retinitis pigmentosa and allied conditions of the fundus, as cases of old choroido-retinitis, with much destruction of the epithelial pigment layer. Patients with this symptom may see fairly well in a good light, in exceptional cases even having normal vision, but when dusk comes on they have to be led about; they also see very badly by artificial light. Night-blindness is frequently accompanied by permanent contraction of the visual fields.

Förster's photometer is a useful instrument if it be desired to record the degree of night-blindness. It consists of a box blackened on the inside. At one end is placed a test-object, such as some reading types. These are illuminated by a light of constant strength admitted through an aperture the size of which can be varied. The patient places one of his eyes to an aperture made for the purpose, and the light opening is gradually enlarged till he can see the test-object. If the apparatus is used under identical conditions the size of the aperture may afford a means of noting and recording the progress of the case. There are, however, considerable individual differences in the light sense.

Bjerrum's test-types, which correspond in size to Snellen's, but are grey on a grey background, afford a ready means of testing the patient's ability to distinguish between grades of illumination, as the contrast between the letters and the background is much reduced. A patient with defective light sense will fail to distinguish the letters from the background, and his visual acuity tested by this method will fall far below that of a normal individual tested in the same way. The value of this test, however, is much weakened by the very wide variations which normal eyes present.

Eyeball, Injuries of.

Injuries of the eyeball are conveniently considered under the following groups:—

1. Injuries by contusion without penetration of the globe.
2. Injuries from penetration without retention of a foreign body.
3. Injuries by penetration with retention of a foreign body.
4. Injuries by strong light, heat, or chemical agents.

One's first duty when consulted by a patient who has sustained any injury of the eye is to ascertain by questioning the manner of its occurrence, and especially by examination the exact nature and extent of the damage done, so that we may be in a position to adopt prompt and effective treatment.

The patient is seated facing a good window light, and the surgeon, placing himself opposite, gently separates the eyelids and exposes the cornea and surrounding sclerotic. If any difficulty is experienced, as sometimes happens with very sensitive persons, a few drops of a 4 per cent solution of cocaine may be first dropped between the lids. In the case of infants and young children it is best to hold the head firmly between the knees, the nurse restraining the legs and arms of the child, which is laid on her lap. The eyelids are now separated by the use of retractors, which are preferable to the fingers with struggling children. It is very seldom necessary to use a general anæsthetic when our object is merely to inspect the eye.

In the examination and treatment of all penetrating injuries of the eye the strictest surgical cleanliness must be adhered to, as septic infection may lead to destruction of the eye, and even to loss of the fellow-eye from sympathetic inflammation. The eye must be well flushed with a 1 in 5000 solution of perchloride of mercury, and on the conclusion of the examination covered with a pad of lint wrung out of the solution. All instruments, as forceps, scissors, speculum, etc., are to be boiled for a few minutes, and laid in a 20 per cent solution of carbolic acid till required. By such measures we can be sure that we do not ourselves infect the wound, but in many cases infection has already taken place at the time of the accident, and in spite of the free use of antiseptics may give rise to septic inflammation of the deeper parts of the eye. If there be present purulent dacryocystitis, evidenced by escape of pus from the canaliculi on pressure over the tear sac, this must be treated at once by slitting up the lower punctum and canaliculus, passing a large probe into the nasal fossa, and syringing the naso-lachrymal canal each day with some strong antiseptic, the most effective for this purpose being a 10 per cent solution of the recently-introduced substance called protargol.[1]

Injuries by Contusion without Penetration of the Globe.—These are produced by light but smart blows on the eye, as from a piece of wood or coal, hard pellet of paper, snowball, small round stone projected from a catapult, cork from a soda-water bottle, etc., which may strike the eyeball directly or through the closed lids. The immediate result of such an accident is to produce slight hyperæmia of the ciliary region with intolerance of light and flow of tears. The pupil soon gets narrowed, and can be dilated by means of atropine only with difficulty. A case of this degree only requires a few days' rest and the use of atropine to recover completely. Some diminution of tension of the eyeball can often be made out, and sometimes this is so pronounced that the cornea and anterior portions of the sclerotic wrinkle on the slightest pressure, and when this is the case the anterior chamber is decidedly shallow. A satisfactory explanation of this phenomenon, which is sometimes permanent, has not yet been given.

A fine greyish cloudiness of the cornea is frequently present, and careful examination by oblique illumination shows that this is situated on the posterior surface, where it is said to be caused by minute elevations and ruptures of Descemet's membrane.

Detachment of some portion of the iris from its ciliary origin, *irido-dialysis*, is a common occurrence; it is accompanied by profuse hæmorrhage, from rupture of the circulus arteriosus major and canal of Schlemm, the blood collecting in a layer at the bottom of the anterior chamber, *hyphœma*. Irido-dialysis is recognised by a crescentic black space corresponding to the position of the detached iris, through which the red fundus-reflex, and even the retinal vessels, may be seen by the ophthalmoscope. The pupil presents a straight or even a convex edge instead of a concave at the site of the lesion. The peripheral border of the detached iris has in a few instances become folded over so as to expose the brown uveal surface, a condition called *anteversion of the iris*. The iris may be detached in two places, but this is extremely rare. In a few instances it has been totally detached, lying free on the floor of the anterior chamber, where it soon becomes of a greyish colour, and shrinks very rapidly.

Multiple ruptures of the pupillary edge of the iris are by no means infrequent, and account for the dilatation of the pupil, *mydriasis*, so often following a blow on the eye. The ruptures appear as small triangular notches in the edge of the iris with their bases directed towards the pupil. They may be seen by focal illumination, but are often best made out by the ophthalmoscopic mirror appearing as breaks in the circular red reflex. Detachments or ruptures of the iris are permanent conditions.

Paralysis of accommodation, *cycloplegia*, recognised by inability to read print close at hand, while the distant sight may be little if at

[1] Obtained from Friedr. Bayer and Company, Elberfeld, Germany.

all impaired, is nearly always associated with mydriasis, but may be present with a pupil of normal size. It is due to paralysis of the ciliary muscle from mechanical injury or hæmorrhage into its substance.

Retroflexion of the iris, first noted by Schmidt in 1804, when as usual affecting only one part of the circumference, gives the appearance of an iridectomy having been done; if the whole of the iris has been thus thrust back the condition resembles total absence of iris, *aniridia*, but is to be distinguished by the ciliary processes not being visible, and careful examination often reveals a very narrow rim of iris at some part of the circumference.

Dislocation of the lens may be partial or complete. The former is recognised by the tremulous state of the iris, *iridodonesis*, at one part, and if combined with rotation on its equatorial plane, by shallowness of the anterior chamber at the opposite side from a pushing forward of the iris by the edge of the lens. If the margin of the lens lies in the pupillary area it is seen as a strongly-marked, dark curved line. In very slight displacement one may require to use the ophthalmoscopic mirror, throwing the light very obliquely into the eye, and dilatation of the pupil is sometimes required, but this sign is seldom if ever absent. Partial dislocation is dependent upon rupture of some portion of the suspensory ligament, but the writer has seen a case where the ciliary body had itself become dragged into the pupillary area, and could be seen with the unruptured fibres of the suspensory ligament stretching between it and the displaced lens. Increase of the refraction, monocular diplopia, and apparent movements of objects looked at, are sometimes present.

The complete degrees of dislocation of the lens, either into the vitreous or anterior chamber, are most apt to occur in elderly people with previous opacity of the lens, or in old damaged eyes, as in such cases the suspensory ligament is easily ruptured.

When the lens lies in the anterior chamber it gives the appearance of a large drop of oil, the edge is very dark and shining, the iris is driven back, and the anterior chamber is deepened. It should always be removed, for inflammation and increase of tension always follow and lead to destruction of the eye. The operation is best done under full anæsthesia by a large flap-like section embracing nearly the lower half of the corneal circumference, and if the lens does not escape spontaneously, or on gentle pressure, it is removed by means of a metal spoon or wire curette. Even with the greatest care we are almost sure to have some loss of vitreous.

A lens dislocated into the vitreous sinks to the floor of the eye, and can be seen by the mirror in this situation as a rounded greyish body, either freely movable, or if some time has elapsed it may be firmly fixed. The iris is tremulous, the anterior chamber deep, and a strong convex lens is required to enable the eye to read print just as is necessary after cataract extraction. In most cases the eye does not remain quiet long, vitreous opacities gather, irido-cyclitis often with increase of tension supervenes, and the eye is ultimately lost, and may even require enucleation. The lens is not absorbed when in the vitreous, and I have found it only slightly diminished in size more than twenty years after the accident. Its removal when in this situation is a matter of very great difficulty, and if the eye remains free from inflammation, and the vision is good, it is best left alone. If it can be coaxed through the pupil into the anterior chamber by depressing the patient's head and shaking it roughly, it may be removed by section as described above, first fixing it by means of a cataract needle thrust through the cornea.

The lens sometimes becomes cataractous as the result of a severe blow on the eye, even without penetration, *concussion cataract*, and Lawford and Collins have by dissection demonstrated in such cases extensive rupture of the posterior lens capsule. In other cases a rupture of the anterior capsule may be present, and as this is situated near the equator its presence may be difficult of recognition.

The *treatment* consists in keeping the pupil dilated, and after complete maturation the cataract is removed by extraction or by discission, followed by the use of the curette. A good visual result may be obtained except when the case is complicated with injury to the deeper structures of the eye.

Hæmorrhage into the vitreous may occur as several small rounded drops; these are from the smaller divisions of the retinal vessels, the actual point of rupture being sometimes visible. They become in time completely absorbed, but very slowly in comparison to hæmorrhage in the anterior chamber. Extensive hæmorrhage can usually be seen as a deep red mass in the lower part of the eye close behind the lens, and probably the source of the blood is from the ciliary body. The vitreous becomes liquefied, as evidenced by the free movement of the broken-up blood-clot, which gradually gets decolorised and assumes the appearance of a flocculent yellowish grey mass. The vision is often reduced to counting fingers or even to perception of light, and improves if at all very slowly.

Rest in bed, the use of atropine twice or thrice each day, combined with the internal administration of ten-grain doses of salicylate of soda or iodide of potash, should be tried.

Rupture of the choroid[1] is not uncommon from a blow on the eye, and is recognised ophthalmoscopically as a white or yellow crescent concentric to, and about two discs diameter from

[1] See also vol. ii. p. 145.

the temporal border of the optic disc. Its greatest width, usually about one-third the diameter of the disc, is at the centre, from which it tapers above and below into a fine streak. The borders of the rupture are more or less pigmented, and minute retinal hæmorrhages are common in the immediate neighbourhood of the lesion. The retinal vessels lie in front of it, and course uninterruptedly across it. It may bifurcate at its upper or lower extension. If seen early, effusion of blood, which always takes place to a greater or lesser degree, prevents a clear view and may render its recognition difficult or impossible. There may be present two, three, or even more concentric ruptures.

Ruptures of this description are explained by the blow causing invagination of the back of the eye by the stem of the optic nerve, the choroid being thus thrown into a fold or series of folds along which this membrane gives way. This phenomenon is often associated with ruptures or detachment of the iris, hyphæma, and dislocation of the lens. The vision, at first much lowered by the effusion of blood, quickly clears up, and may reach a normal standard if the region of the macula lutea is not involved. The retina is very rarely torn, but may become detached long afterwards during cicatrisation. Atrophy of the optic disc sometimes occurs, and dotted pigmentary degeneration of the macula, *Haab's macular disease*, is often present. In a small percentage of cases the rupture is situated at the nasal side of the disc.

We sometimes get an irregular map-like area of yellowish discoloration of the choroid commencing near the disc and spreading out like a fan towards the periphery either at the temporal or nasal side of the eye, with pigment strewn over its surfaces and penetrating into the retina. Such cases have been described by Siegrist, who believes the condition is due to rupture of one or other long ciliary artery, as precisely similar changes were shown by Wagenmann to follow experimental division of these vessels.

The term *commotio retinæ* some five-and-twenty years ago was employed to designate all cases of defective vision following a blow on the eye if gross ophthalmoscopic changes, especially atrophy of the disc, were absent, but since Berlin's observations and experiments the term has become much more restricted in its application. Berlin, in many cases of blows on the eye, found a diffuse greyish clouding at the posterior pole which came on within an hour of the accident, gradually increased in size and density, becoming of a milky white colour and covering an area ten or a dozen times the size of the disc, against which the retinal vessels stood out very dark and distinct, but showed no elevation.

These changes reached their maximum in twenty-four to thirty hours, and had usually disappeared by the third or even the second day, with complete or nearly complete restoration of the vision, which at first had fallen to one-third or a quarter of the normal standard. By his experiments on animals he found a layer of blood between the choroid and sclerotic corresponding in position to the retinal œdema, which latter he considered was due to imbibition of serum from the subchoroidal effusion of blood.

This condition being so transitory is very readily overlooked, especially as the intolerance of light set up by the blow renders it very difficult to make a proper ophthalmoscopic examination. The clinical features of this affection have been confirmed by all subsequent writers, among whom may be mentioned Leber, Hirschberg, Schmidt-Rimpler, Nettleship, Knapp, and Herdegen.

As to the exact cause of the retinal clouding which several have tried to elucidate by experiments on animals, opinions are almost as numerous as the communications. Hirschberg and Ostwalt say it is due to a paralytic widening of the retinal vessels followed by increased permeability of their walls, the œdema thus spreading from the inner to the outer layers of the retina, and not, as Berlin thinks, in a reverse direction, and this view is accepted by Siegfried and Linde.

Makrocki thinks it is due to a molecular change in the nerve-fibre layer. Denig believes there are two factors at work, the fluids of the vitreous being driven into the retina and a transudation from the choroidal vessels collecting between the rods and cones.

Schirmer found hæmorrhage between the retina and choroid, which being constantly present he regards as the cause of the retinal clouding, while finally Bäck thinks it is due to a fine fibro-nuclear layer of exudation between the retina and choroid.

It is impossible to reconcile these discrepant views, and personally I think Berlin's explanation as satisfactory as any. We must keep the pupil dilated with atropine, forbid the use of the eyes, and make the patient wear dark smoked goggles.

Traumatic neurosis may very rarely follow injury of the eye as after injury of other parts of the body, and is shown by amblyopia with peripheral contraction of the field of vision, blepharospasm, cutaneous anæsthesia, achromatopsia, etc.

Injuries from Penetration without Retention of a Foreign Body.—The cases under this heading are of much greater interest to the practical surgeon than the preceding group, as they demand more energetic and varied treatment, and derive especial importance from the fact that the sound eye is often in danger from sympathetic disease, necessitating removal of the injured eye.

A heavy blow or thrust with some blunt object, or a fall on the same, may burst the

globe, which gives way, not at the point of impact, but at some distance from it. This *indirect rupture* is in adults almost invariably in the sclerotic, in young persons it may be in the cornea; it is most frequently directly upwards, or upwards and inwards, and is concentric to and about 2 to 3 mm. from the corneal margin. The coats of the eye give way from within outwards in contrast to what takes place in direct ruptures, so that septic infection is not at all liable to occur; the eye may shrink, but does not suppurate. If the conjunctiva escapes, the rupture is recognised by a dark curved line, the tension is greatly diminished, the eye full of blood, and vision is usually reduced to perception of light. If, as is often the case, there be extensive effusion of blood under the conjunctiva, the actual rupture cannot be seen, and its occurrence can only be inferred from the history and symptoms, especially the softness of the eyeball. Some of the contents of the eye are often forced through the gap in the sclerotic, very often the lens is dislocated under the conjunctiva, and shows as a rounded, semi-translucent swelling, or if the conjunctiva is torn it may escape altogether. The iris may retain its position, but usually the segment corresponding to the rupture is forced backwards into the wound, giving the appearance of a coloboma.

Indirect ruptures may very rarely be situated in the posterior segment. Mules records such a case in a lad who was struck with a brick, where the sclerotic around the optic nerve formed a hernial protrusion backwards, associated with a typical rupture of the choroid. The lens has been found in Tenon's capsule from scleral rupture unusually far back, as in cases of Müller and of Schlodtmann. The iris may escape bodily with or without the lens, or it may be caught and retained under the conjunctiva as in a case under my charge, where it showed as a dark, irregular, and slightly-raised patch from which the pigment wandered, so that in a few weeks the entire ciliary region became covered with pigment granules. See cases by Hirsch, Ahlstrom, Knapp, and Gayet.

Treatment of Indirect Rupture.—If the rupture is large, the conjunctiva torn, and there has been much loss of vitreous, causing collapse of the globe, excision of the eye should be done without delay. If there is some sight and no marked collapse of the globe, an attempt should be made to save the eye.

Any protruding uveal substance is picked up with iris forceps and excised with scissors, and the edges of the conjunctiva are brought together by a few fine silk sutures. It is a good plan, as recommended by Berry, to excise some conjunctiva at one lip of the wound, so that when the stitches are applied the wound becomes quite subconjunctival and removed from the line of sutures. Cases of subconjunctival dislocation of the lens often do remarkably well without any surgical interference, the blood clears away, the scleral wound unites solidly without any bulging or sinking, and fine print can be read with a strong convex lens. Mules records in vol. vii. *Trans. Ophth. Socy.* a case where both eyes made a good recovery from an injury of this nature from the blow of a fist. This favourable course probably occurs in those cases where the lens is thrust quite through the lips of the wound. Some recommend removal of the lens in all cases at an early date. Personally, if the eye is doing well, I prefer to leave it alone for some weeks or even months till the wound is quite healed, and then open the conjunctival covering and clear out the lens. If, on the other hand, the lens with some portion of the uveal tract is jammed in the wound, the healing process is very tedious, plastic uveitis comes on, and may lead to sympathetic disease, as in the case from which Fig. 1 was drawn, a woman who repeatedly refused to have the eye removed.

Under these conditions the lens and any portion of uvea protruding should be removed and the eye dealt with as described above. If in spite of this the eye does not quieten down in a month it must be excised.

Direct ruptures and wounds of the eyeball may take place from the thrust of a cow's horn, a blow from the branch of a tree, piece of wood, as in chopping up fuel, "tip-cat," stone from catapult, flying shuttle, police baton, or from a stab from a rapier, knife, nail, fork, iron table-fork, or pitchfork, or from the bursting of a steam gauge or soda-water bottle, etc.

In the most severe cases, especially when combined with extensive injury of the lids and adjacent parts, the patient may show general symptoms of collapse, and Fromaget records a case of tetanus, with death on the eighteenth day after the injury, and later with Cabannes another case with a fatal issue about the tenth day. See also case by my colleague Edward Roberts (*Lancet*, 1891).

The wound may be confined to the cornea or the sclerotic, or it may implicate both. Speaking generally, corneal wounds are not so liable to be followed by severe destructive inflammation as are those in the sclerotic, and as pointed out by Berry this is due to the gush of aqueous at the moment of the accident washing away any septic matter, and to the lips of the wound not being kept apart by prolapse of the inner coat of the eye or the vitreous. The gravity of the case depends more largely upon the depth of the wound than upon its external measurements, and from this it follows that, as in other parts of the body, stabs and punctures are more destructive than incised wounds. In Figs. 2 and 3 we see corneal stabs followed by suppurative hyalitis, with papillo-retinitis in one and detachment of the retina in the other. In neither eye was there the slightest implication of the choroid,

although clinically such cases are very often, but, as pointed out by Schöbl, quite erroneously termed irido-choroiditis. On excision for corneal stab wound I have found the coats of the eye transfixed in the posterior segment. Exploration of the wound by means of a probe is here of course quite inadmissible, and we must judge of its depth by the state of the vision, the opacity of the lens, the presence or absence of vitreous in the wound, and of blood or exudation in the vitreous.

Corneal wounds are usually linear and radial, but may be flap-like or triangular, T-shaped, Y-shaped, or irregular; the edges may lie in perfect apposition or overlap, or be kept apart by prolapsed iris, and there may be greyish infiltration for a little distance around. A two-pronged fork will often give rise to a double puncture. The capsule of the lens is very often injured in corneal wounds, and this is always followed by traumatic cataract, which if the capsular wound is large undergoes spontaneous absorption. When the wound in the capsule is a mere puncture it may become sealed up, and the progress of the cataract is arrested.

Scleral wounds are particularly serious when situated in the ciliary region, which corresponds to the sclerotic for one-quarter of an inch from the clear cornea, and has well been termed the "dangerous zone." Such wounds often lead to shrinking of the injured eye and sympathetic disease of the other. Unfortunately this is the region of the sclerotic most exposed to injury, and wounds farther back are comparatively uncommon.

One or more *eyelashes* not infrequently get drawn into the anterior chamber in penetrating wounds of the cornea, and a great many cases are on record. See paper by Landmann (Graefe's *Archives*, xxviii. 2) with full references. They usually lie flat on the iris, but may have one end fast in the iris and the other free in the aqueous; they are rarely seen till the eye has recovered from the accident, and even then are readily overlooked. If some of the cells of the root sheath accompany the lash, these proliferate and form an epithelial *pearl growth;* see Rockliffe, and Cross and Treacher Collins, the latter with full list of cases. These pearl growths take nearly twelve months to attain the size of a split pea, they resemble a little mass of camphor with the lash protruding from it, and should be removed.

Apart from the occasional occurrence of these growths, little if any harm results to the eye from intrusion of a cilium; the writer had a case where a lash had remained in the anterior chamber for twenty years, the lens was absorbed, and the finest type could be read with a suitable glass. Schwartz describes cases in which lashes had given rise to the occurrence of giant cells. Penetrating corneal wounds also in some cases give rise to the formation of *serous cysts* in the angle of the anterior chamber, and according to Eversbusch these are due to inversion of the ligamentum pectinatum and endothelial lining of the iris angle, with accumulation of aqueous in the pouch thus produced. They are of very slow growth, but if not removed will certainly lead to destruction of the eye from increased tension.

Treatment of direct Penetrating Wounds.—Wounds in the ciliary region with loss of vitreous, and with or without injury of the lens, require excision of the eyeball, and this should be done before the onset of panophthalmitis, as fatal purulent meningitis sometimes attends the operation in this condition. Wounds of the sclerotic behind the dangerous zone may be treated by conjunctival sutures as noted on page 211, and a good result as far as appearance is often obtained, but detachment of the retina and total loss of sight is very common in all scleral wounds, and unless there is a prospect of saving some useful amount of sight, primary enucleation is to be preferred. Generally speaking, if the eye has not quietened down after a month, or at latest six weeks after the injury, further delay is unjustifiable, and excision is called for, as sympathetic disease is apt to come on at this time. Clean cut wounds of the cornea without prolapse of the iris heal up quickly and satisfactorily by the use of a compress wrung out of the perchloride lotion, and kept in position by a roller bandage. When in the course of a few days the anterior has reformed, atropine is used thrice a day till all injection of the eye has disappeared. When prolapse of the iris has taken place no attempt should be made to reduce it, as it is difficult to do, and may introduce septic matter into the eye. In recent cases there is no difficulty in seizing the prolapsed iris with curved iris forceps, laid with the blades parallel to the corneal wound, drawing it well forward and snipping it off with scissors. This will have the effect of freeing the iris at both lips of the wound, and leaving a coloboma almost as regular as after an ordinary iridectomy. If some days have elapsed we must endeavour by the use of a fine silver probe or spatula to break down and separate the adhesions of the iris along the whole extent of the corneal wound and at both lips, and then draw out and excise the iris. The recovery largely depends upon the successful accomplishment of this procedure. If the lens has been wounded it is best left alone, to be spontaneously absorbed or dealt with as mentioned on page 211. If purulent hyalitis supervene, indicated by the ominous yellow reflex behind the lens, the eye is doomed and should be excised, but if panophthalmitis is present it is safer to perform the operation of *evisceration*, *i.e.* removal of the cornea and surrounding ring of sclerotic, and clearing out all the contents of the eyeball with a Volkmann's spoon.

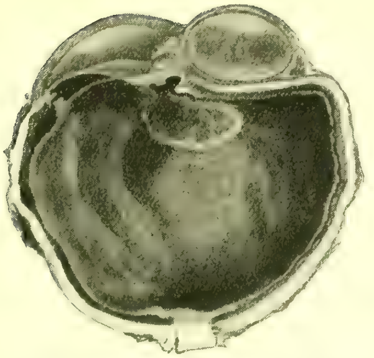


FIG. 1.—Sub-conjunctival dislocation of lens causing sympathetic disease (unusual result). The edges of the ciliary rupture are kept apart by the lens. The entire uveal tract is uniformly and markedly thickened to several times its normal diameter, a condition characteristic of sympathetic disease.

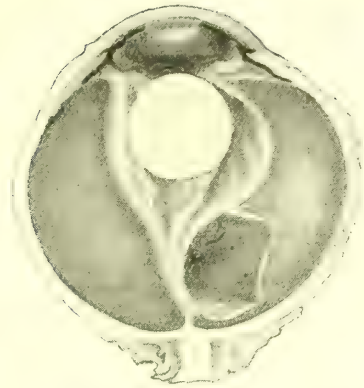


FIG. 2.—Puncture wound of cornea causing suppurative hyalitis and detachment of retina with cystic space in the posterior portion of the retina.

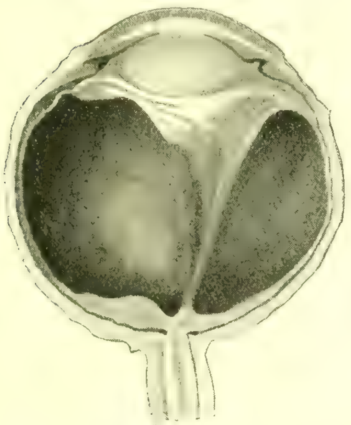


FIG. 3.—Puncture wound of cornea causing purulent cyclitis, the exudation in ciliary region and posterior surface of lens stretching towards the back of the eye, where well-marked papillo-retinitis is seen.

FIG. 4.—Old lost eye—a mere stump—which caused after a long period of immunity destruction of fellow eye from sympathetic disease.

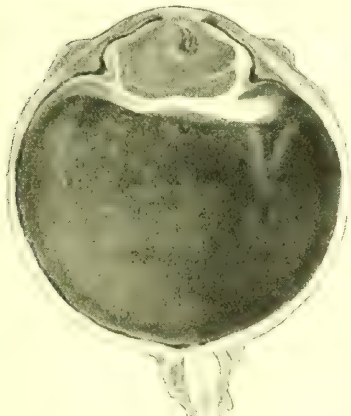


FIG. 5.—Foreign body in lens, commencing purulent infiltration of eye. Septic infection must have taken place at accident, as foreign bodies are usually well tolerated in this situation.

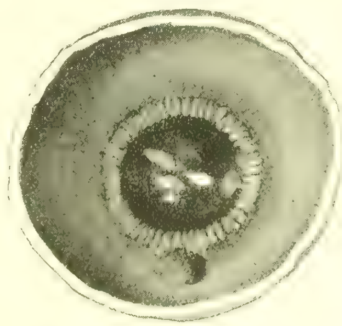


FIG. 6.—Piece of metal stuck fast in lower part of ciliary body, a common situation which is reached by a rebound from the back of the eye.

These Figures are from drawings by Dr. W. E. Fothergill of specimens in the possession of the author, and enlarged to two diameters.

Corneal wounds which reach a short distance into the sclerotic are quite as liable to produce sympathetic disease as wounds confined to the sclerotic, but detachment of the retina and other changes in the eye are not so apt to occur, and good results are sometimes obtained by careful treatment. Snellen detaches the conjunctiva freely at each lip of the wound, scrapes the epithelium from the adjacent parts of the cornea, and then by sutures endeavours to render the entire wound subconjunctival.

All lost, shrunken, and tender eyes should be excised; Fig. 4 is an example of a mere stump of an eye which caused sympathetic disease.

Enucleation of the eyeball, also called excision and extirpation, should always, when possible, be done under a general anæsthetic, and is performed in the following manner:—

The eyelids are kept apart by a spring speculum, the conjunctiva is seized with fixation forceps quite close to the upper edge of the cornea and freely separated with curved, blunt-pointed scissors as far back as the insertion of the rectus tendon. The rest of the conjunctiva all round the cornea is then dealt with in a similar manner. A squint hook is inserted under the tendon of the superior rectus muscle, which is then divided with the scissors as close to the sclerotic as possible; the tendons of the internal inferior and external recti are divided in the same way. The speculum having been unscrewed, but still in position, is pressed back firmly into the orbit, when the eye will spring forward, and is lightly held between the forefinger and thumb of the left hand. A strong, curved, blunt-pointed pair of scissors, with the blades closed and their concavity looking forward, are then pushed from the temporal side of the eye back into the orbit till the optic nerve is felt, which is then divided by one snip. The tendons of the superior and inferior oblique muscle are now divided and the eyeball is removed. The cavity is well flushed with perchloride lotion, and when the bleeding has stopped the edges of the conjunctiva may be brought together by two or three sutures, but these are often dispensed with. I much prefer the plan used more than forty years ago by Bowman and Critchett, of running a single thread out and in along the margins of the conjunctival gap and drawing together but *not* tying the ends. On the third day the thread is easily withdrawn by pulling on one end. The eyelids are inverted by a thick pad of lint placed on their outer surface, which is kept in position by a roller bandage.

An artificial eye may be worn in a month after the operation; it is a thin shell of glass made to resemble as closely as possible the front part of the sound eye. Snellen has recently had glass eyes made by Messrs. Müller of Wiesbaden[1] in the shape of hollow globes, more or less flattened at the posterior pole; they are said to be more comfortable on account of the rounded edge, and also look better, as they do not have the sunken appearance so often noticeable with the old form. The old form is still the most suitable when worn over a shrunken globe, or where evisceration or Mules' operation of the artificial vitreous has been done in place of enucleation.

1 Can be obtained from Krohne and Sesemann, 37 Duke Street, Manchester Square, London, W.

Injuries by Penetration, with Retention of a Foreign Body in the Eye.—Small particles of iron or copper, splinters of glass, porcelain, or wood, small shot, and other small, hard bodies striking the eye with great velocity, penetrate the cornea or sclerotic, and come to rest in any part of the eye, giving rise to a very serious condition. The wound of entrance being small and the fragment being as a rule aseptic, the gravity of the case depends upon the presence of the foreign body, and generally speaking the eye is doomed from repeated attacks of inflammation unless we succeed in removing this. The evil effects of the foreign body are due to the mechanical and chemical irritation which it sets up, and vary in intensity with its material size and position in the eye. Large, irregular, sharp-angled bodies do more harm than small, smooth ones; copper is much more obnoxious than iron or steel, and lead is certainly better tolerated than either of the latter.

Foreign bodies in the aqueous chambers are much less dangerous and more easy of removal than those in the vitreous chamber, and we shall consider the two classes of cases separately.

Foreign bodies in the aqueous chambers almost invariably gain entrance by perforating the cornea; in the few exceptions, usually in gunshot accidents and explosions, the foreign body goes through the lid and outer part of the sclerotic, and reaches the lens or iris or anterior chamber from behind. The corneal wound being very small and rapidly produced there is no prolapse of the iris, the aqueous is often completely retained, and even if it escapes and the anterior chamber is abolished, it is quickly restored. By careful focal illumination the foreign body can be detected in the iris, lens, or more rarely, free in the anterior chamber, but in the latter case, if it be very small it can be quite hidden in the sulcus between the base of the iris and the opaque limbus, or if it lies behind the iris in the posterior aqueous chamber, its presence may only be suspected by a bulging of the iris, with or without a tear in that structure. Blood may be present in small quantity, either as a layer on the floor of the anterior chamber or more frequently as a clot on the anterior surface of the iris. The pain is often very slight, and persists only for a very brief time. The vision at first is but slightly if at all diminished unless the lens has been wounded.

Treatment.—The removal of a foreign body

from the aqueous chambers is not to be lightly undertaken by any one who has not had some operative experience on the eye, for a delay of a few days will probably do less harm than the attempt at removal by a novice. Children must be deeply anæsthetised, but cocaine is preferable in adults. The instruments required must be at hand, and should comprise speculum, fixation forceps, and scissors, Graefe's knife and keratome, curved iris forceps, also small curved forceps with broadish ribbed blades, a curette, and a Daviel's spoon. Very good daylight is necessary, or, failing this, an electric focus lamp. The pupil should not be dilated with atropine; it is often advantageous to contract it with eserine, which permits of a much better view of any substance in the anterior chamber and renders injury of the lens less liable.

The corneal section should be made with a Graefe's knife rather than with a keratome, so as to produce a flap, and this should not be too small. If the foreign body is free in the anterior chamber it may, by a piece of luck, at once escape with the rush of aqueous, but in most cases it requires to be laid hold of and removed. Small shot and other round bodies are best dealt with by the Daviel's spoon, scales and chips of metal or glass or other hard, smooth substances should be seized with the ribbed forceps, while fragments of wood and other softish material are readily removed by means of the iris forceps. Foreign bodies on the iris, if not deeply embedded, may, as recommended by Knapp, be removed by a small blunt hook or a curette without excising iris, but if buried in the iris it is best to seize the foreign body and iris together, draw them out of the eye, and cut off with scissors.

Foreign bodies in the lens are better tolerated than in any other part of the eye, and may be left till complete cataract is produced, which is then extracted together with the foreign body.

A foreign body *behind the iris* may sometimes be seen protruding in the pupil, but at other times its presence is shown only by a bulging of the iris. Its removal from this position always necessitates an iridectomy. The lens is usually wounded at the time of the accident and must be dealt with later.

Foreign Bodies in the Vitreous Chamber.—These not infrequently penetrate the coats of the eye and lodge in the vitreous, reaching this locality by going (1) through the cornea and lens; (2) through the cornea, iris, and lens; (3) through the cornea, iris, and zonule; or (4) through the sclerotic and underlying coats of the eye. In some cases the lid, usually the upper, is also perforated. The diagnosis of such an occurrence is often a matter of considerable difficulty, and frequently we can only arrive at a probability that a foreign body has entered the eye.

The first thing we do is to search for an opening or scar in the anterior part of the eye. If this lies in the cornea there is seldom any difficulty in detecting it, but if very small and in the sclerotic it is more likely to be overlooked from the presence of blood in, or thickening of, the conjunctiva. A good light and the use of a magnifying lens may here be necessary.

The patient's testimony, further than that he has received an injury, is seldom to be trusted. Associating as he does the idea of "something in the eye" with the occurrence of constant pain, he usually assures us that there is nothing in the eye, and even goes the length of declaring that he saw the piece of metal or other substance that struck the eye, when we have undoubted evidence that it is actually in the eye.

Having made out the point of entrance, let us assume that this is in the cornea and about its centre—we find a mark on the anterior and on the posterior capsule, with a connecting streak running through the lens. If more eccentric a hole in the iris will indicate the course of the foreign body, which in this case may reach the vitreous by going through the suspensory ligament in the circumlental space.

By the above signs we may be quite sure that a foreign body has penetrated into the deep parts of the eye, but we must bear in mind that this may have gone through the globe to be lodged in the orbit. I have seen this on several occasions with small, sharp pieces of steel and lead pellets, but have never (before the use of Röntgen rays) diagnosed the condition before enucleation of the eye. I had a case in which a sharp, sword-like piece of steel over an inch in length penetrated the cornea and went clear through the eye, one end becoming firmly fixed in the bony wall of the orbit, the eye being impaled on the other end. The foreign body may reach the vitreous directly by going through the sclerotic, in which case careful probing may be required to ascertain if the whole thickness of the sclerotic has been perforated. An opaque streak in the vitreous, one end corresponding to the wound in the sclerotic, is often visible and indicates the track taken. When the foreign body has taken this route we are much more likely to have hæmorrhage in the vitreous than when it has gone through the lens, rendering its detection more difficult; but in the latter case opacity of the lens takes place and, if advanced, may prevent our making out the foreign body.

The foreign body may get entangled in the coats of the eye at the point of entrance; it may be free in the vitreous, or be suspended there in a blood-clot or string of lymph. It may traverse the vitreous and become embedded in the retina; it may rebound from the back of the eye and come to rest on the posterior part of the ciliary body below, as in Fig. 6; or

finally, as already mentioned, it may go through the globe into the orbit.

If the media are fairly clear, we may with the ophthalmoscope see the foreign body as a dark object with glistening margins in the vitreous or embedded in the fundus. Bubbles of air are sometimes met with in the vitreous, and are strong but not absolute proof of the presence of a foreign body; they are round, often multiple, glisten at the centre, and have a dark, sharply-defined border, by which characters they can easily be distinguished from solid substances. They always disappear in from one to two days.

Choroidal plaques, often very bright and silvery looking, give us the impression of a plate of metal lying on the fundus, but can be distinguished by their shining all over and not merely at the margins. When a foreign body has rebounded from the back of the eye we find a spot where the sclerotic is exposed, with some hæmorrhage around it, and when present this affords us valuable information as to the nature of the accident.

Another piece of corroborative evidence is the presence of a finely speckled appearance of the macula, which is common even when the foreign body lies at a considerable distance from this part of the eye.

Chemically indifferent and smooth objects, such as a piece of glass, a lead pellet, or a portion of highly-polished wire, may remain a long time in the vitreous without causing any inflammation; but a fragment of iron with roughened surface soon gives rise to inflammation, and becomes completely encapsuled in lymph in about a fortnight, after which the inflammation subsides, but is very apt to recur.

Rusty discoloration of the iris, lens, and other tissues takes place from lengthy retention of a piece of iron, and in such cases is of some diagnostic service.

Removal of Foreign Bodies from the Vitreous Chamber.—When the body is not magnetisable our chance of successfully removing it is so very small that this should be attempted only if we can actually see it, and it happens to lie near the equator, either in the superficial layers of the vitreous or in contact with the coats of the eye. Under these circumstances an incision is made by a lance-thrust, when gentle pressure may cause the foreign body to escape with a slight prolapse of vitreous, or a forceps may be introduced and the foreign body seized and removed. Unfortunately, however, it frequently happens that our attempts are fruitless: the vitreous escapes, but the foreign body remains in the eye.

Before undertaking the operation, it is advisable to get the patient's permission to remove the eye at once if we should not succeed in extracting the foreign body.

If it is a piece of iron or steel that has entered the eye, and, fortunately, 75 per cent of all cases are of this nature, our chances of success by the use of the electro-magnet, first devised and employed systematically by Hirschberg in 1879, are very much greater. Hirschberg's instrument, consisting of a cylinder of soft iron surrounded by wire, connected with a battery, and tapped at one end to hold different-sized probes, is convenient and inexpensive. Similar instruments have been devised by Snell, M'Hardy, Froehlich, Jany, and others.

We are much more likely to succeed if the operation be undertaken within a day, or not later than two, after the accident, before it becomes covered with lymph, and if it is not embedded in the coats of the eye. When an operation is considered advisable it should be done at once.

If the wound of entrance is in the sclerotic, it should be enlarged and the electric needle thrust into the vitreous towards the metal if seen, or in the direction in which it is supposed to lie. If successful, a "click" is both heard and felt, and on slowly withdrawing the instrument the foreign body will be found attached. The conjunctiva is then sutured over the wound in the sclerotic.

If the foreign body has gained the vitreous by going through the cornea, and the lens is opaque, we should extract the cataract by a lower flap, and then make for the foreign body by passing the electric needle through the operation wound. If the lens has not been wounded our best plan is to dissect up a conjunctival flap, about the equator, towards the outer side of the inferior rectus, and with a Graefe's knife make an incision five or six millimetres in length in a sagittal direction. The knife should be thrust deeply into the vitreous to reach the foreign body, if possible, which renders the use of the magnet much more effective.

If we are in doubt as to the presence of a piece of iron or steel, the use of the sideroscope will, it is said, tell us for certain by the "dip" of the balanced needle which is enclosed in a glass tube, and by placing the tube on different parts of the front of the eye we can judge the position of the foreign body with sufficient accuracy to decide where we should make our scleral incision.

Haab's giant magnet,[1] a large cylindrical-shaped instrument weighing 30 kg., with blunt conical extremities, is of great service. If the patient be placed with his cornea close to or in contact with one of the poles, the occurrence of sharp pain tells us of the presence of a fragment of iron or steel which is drawn from any part of the interior of the eye, either into the anterior chamber or through the circumlental space to the back of the iris, which is seen to bulge.

[1] Obtained from the maker, Jacob Rieter et Cie, Winterthur, Switzerland. Cost 450 frs.

The removal is effected by a corneal section and the use of forceps or Hirschberg's magnet. The great advantage of this powerful instrument is in dealing with *small* fragments which cannot be seen, or which have lain a long time in the eye.

The use of Röntgen rays had been practically of no use in ophthalmic surgery till Mackenzie Davidson worked out a plan which enables us not only to detect foreign bodies in the eyeball or orbit, but to estimate their exact size and position.[1]

The method is essentially as follows:—

1. The patient is seated with the photographic plate in contact with that side of his head which corresponds to the injured eye, the Crookes tube is on the opposite side of the head, 30 cm. from the plate, and exactly opposite the point of crossing of two wires stretched across the frame holding the plate, the one vertical and the other horizontal.

2. The wires are inked so as to mark the patient's temple, a small piece of lead wire is made to touch and mark the lower eyelid at a point which bears a definite relation to some point of the eyeball, such as a scar, etc.

3. Two skiagrams are taken, one upon the plate with the Crookes tube 3 cm. behind, and the other upon a celluloid film 3 cm. in front of the point of crossing of the wires.

4. The two negatives, the celluloid one being uppermost, are then placed on the "localiser"—a glass horizontal stage with a reflector underneath.

The adjustments are now repeated with a fine silk thread going from the anode in its first position to the corresponding image of the foreign body, and another thread connecting the anode in its second position to the corresponding image. The point where the threads intersect fixes the position in space of the foreign body in relation to the image of the cross wires.

Radiography has already been of signal service, it tells us for certain if the foreign body has gone through the eyeball into the orbit, a thing we could never be sure of before the use of this method, and when this is the case we may usually with safety retain the eye.

Injuries by Strong Light, Heat, or Chemical Agents.—Momentary contact of a gas flame, hot curling-tongs, or cigar ash, produces a superficial burn of the cornea, shown by a limited area of milk-white opacity of the epithelium, which, being thrown off, exposes the nerve-ends, and gives rise to severe smarting pain, continuing till the epithelial covering is restored.

[1] The apparatus is expensive, the whole outfit costing about £80. Good electric coils may be got from Apps, 433 Strand; Crookes' tubes from A. C. Cossar, 67 Farringdon Road, E.C.; and the Mackenzie Davidson localiser is made by Muirhead and Company, Westminster.

Healing is brought about in a few days by the use of a bandage and the instillation of atropine. Burns by molten lead are chiefly remarkable for the comparatively slight injury done, probably on account of the parts being protected by a layer of vapour; the metal forms a more or less complete cast of the conjunctival sac, and, when removed, one is astonished to find only a superficial destruction affecting the corneal and conjunctival epithelium. Molten iron, on account of its much higher temperature,—nearly four times as great as molten lead,—produces a far more serious burn; the cornea and sclerotic at the affected part assume a dense white appearance from coagulation of the albumin in the tissues, often with the edges of a yellow colour. The sensibility is much impaired or quite lost, indicating necrosis of the parts, which by secondary inflammation around are later thrown off as a slough exposing the vitreous, and leading to total loss and shrinking of the eye, or in less severe cases to deep ulceration of the cornea and sclerotic, with iritis and adhesions between the iris and lens capsule.

Strong mineral acids, or alkalies—as potash, soda, ammonia, and lime—sometimes get in the eye, but we shall refer further only to lime burns, as these are very common, often serious, and demand prompt attention. The lime is almost invariably in a slaked or partially slaked form, and sometimes mixed with sand; it has a most deleterious effect on the cornea, and if not quickly removed from the conjunctival sac, which can only be done by proper medical aid, it continues to act upon and destroy the tissues. In the least severe cases the cornea is but slightly clouded, in moderately severe cases the cornea becomes like ground glass, and in the worst there is a dense white porcelain-like opacity with loss of sensibility. Ulceration often with perforation takes place leading to adherent leucoma. The ocular and palpebral conjunctivæ are always involved and become adherent, giving rise to a condition called "symblepharon," which is called "anterior" when a probe can be passed under it, and "posterior" when the adhesion takes place right back to the reflected conjunctiva. "Pterygium" also often occurs during the healing process by a fold of the ocular conjunctiva becoming adherent to a corneal ulcer.

Treatment.—All particles of iron, lead, lime, or other foreign substances are to be picked off with forceps, or they may require to be dug out with a spud. The use of a 5 per cent cocaine ointment will render this process less painful. Particles of lime are especially hard to remove; the lids are everted and gently rubbed with pieces of lint soaked in oil or vaseline, and particular attention must be paid to the retrotarsal fold.

In lime burns we must avoid the use of

water, a strong syrupy solution of sugar is the best thing to employ at first, or failing that, we may use vinegar. No bandage should be applied, as it favours the formation of symblepharon, and once or twice each day a probe is used to prevent or break down adhesions. A solution of atropine in olive oil, four grains to the ounce, should be freely applied several times a day with a brush. The lids are often hard, infiltrated, and swollen, rendering it very difficult or impossible to fully evert the upper lid, and hot fomentations are here grateful and useful. If the conjunctival surfaces are burnt back to the sulcus, it is quite impossible to prevent union, and one must rely upon some plastic operation later on to cure or alleviate the symblepharon.

In the case of burns by acids we use a 1 per cent solution of carbonate of soda to flush out the conjunctival sac, and in the case of alkalies warm milk is the best thing to use.

In all severe burns during the separation of the slough a warm solution of corrosive sublimate (1 in 5000) must be freely used, and if the eye is already lost and the pain is severe hot antiseptic poultices do good.

In the most severe cases of burn with molten iron or lime, with deep sloughing and consequent perforation in the corneo-scleral region, one may have to enucleate the eye, as sympathetic disease is liable to take place.

After watching a solar eclipse without the precaution of using very dark spectacles, damage may accrue to the delicate structure of the retina, shown in the least severe cases by the persistence for hours or days of an after-image of the sun in the form of a golden disc, most noticeable in the dark. No changes can be detected in the eye, and confinement to a darkened room for a few days will effect a complete cure. In more severe cases we get a decided reduction of sight, the presence of a positive scotoma in the centre of the field of vision, and a "trembling" of objects looked at such as is produced by hot air. The ophthalmoscope shows at the macula a white, burnt-like spot which disappears in a few days, leaving the macula more sharply defined and of a darker red than natural, often with one or two minute greyish flecks; the foveal reflex is abolished. These changes are without doubt the direct and immediate result of the light of the sun focussed on the fundus, and in some recorded cases the grey fleck on the macula has taken the form of a half-moon, exactly like the sun at the beginning of an eclipse. In the course of a week or ten days central vision has usually considerably improved, but some amount of defect remains permanently, and in two of Haab's cases vision was only one-third and one-fifth of normal respectively after so long a period as ten years.

The ill effects of the electric light are seen in those whose duty it is to adjust strong arc lights of say 2000 candle-power. The first result of incautious exposure of the eyes is a dazzling and partial blindness, everything looked at having a yellowish cast; this is followed in six to nine hours by intolerable smarting pain and a feeling of grit in the eyes, profuse lachrymation, swelling, and redness of the lids, chemosis of the ocular conjunctiva, hyperæmia and even actual inflammation of the iris, with the formation of adhesions to the lens capsule.

Iced compresses are the best thing to use at the start, followed by the instillation of a mixture of cocaine and atropine when the crisis is reached. Complete recovery always takes place.

The effects of lightning on the eyes are very numerous, and comprise superficial burns of the lids and cornea, blepharospasm and intolerance of light, conjunctival injection and corneal cloudiness, iritis and cyclitis, cataract, spasm, or paralysis of accommodation, mydriasis or myosis, rupture and detachment of choroid, vitreous opacities, retinal hyperæsthesia, and retinal hæmorrhages, pallor or hyperæmia of disc, neuritis and atrophy, paralysis of muscles and ptosis. Some of these effects are mechanical in their origin, others are thermic, chemical, or catalytic. The most frequent and important are clouding of the cornea and opacity of the lens; in a case reported by Silex both conditions were present, but partially cleared away in eleven months; and in Denig's case the double parenchymatous keratitis totally disappeared in four weeks.

The cataract is usually bilateral, may be complete or incomplete, and in the latter case may rarely retrogress. The origin of the cataract is probably not alike in every case; it has been regarded as a "concussion cataract" by some, from coagulation of the albumin, as secondary to injury of the ciliary body, and on experimental evidence Hess believes it is due to destruction of the capsular epithelium.

Extraction is followed by satisfactory healing, but the visual result is often poor on account of coincident injury of the uveal tract or optic nerve.

Eyelashes. *See* Eyelids, Affections of (*Trichiasis*, etc.).

Eyelids, Affections of.

Anatomy, etc.—The eyelids are *developed from* two folds of epiblast, which grow together from either side of the ocular cleft until they meet in front of the globe to form a closed cavity, the conjunctival sac; and from the mesoblast which extends into these folds are derived the non-epithelial tissues of the lids — the tarsus, muscles, fasciæ, etc. The line of union, where normally the folds meet, separates again before birth to form the palpebral fissure; and by the defective union or separation of these folds various congenital deformities of the lids are produced.

When the eyes are closed the palpebral fissure forms a tear-tight slit between the lids, which varies in length and direction in different people. Its ends, where the lids join together, are the outer and inner canthi, and its margins are formed by the edges of the tarsal cartilages. The apparent size and shape of the eyes, when the lids are open, depend upon the length and direction of the palpebral fissure, which allows more or less of the cornea and sclerotic to be exposed.

The skin of the lids is very thin, and is loosely attached to the subjacent tissues, except over the area of the upper tarsus, where it adheres a little more firmly. At the margin of the palpebral fissure the skin, where it merges into the palpebral conjunctiva, is intimately attached to the edge of the tarsus.

The *subcutaneous cellular tissue*, which after infancy is devoid of fat, is so loose and delicate that it admits of great swelling of the lids, which may occur very rapidly.

The *tarsal cartilages* are two thin semilunar-shaped plates of fibrous tissue, wedge-shaped in vertical section; the thick edge of the tarsus forms the margin of the palpebral fissure, and the thin edge is attached to the orbital margin by the palpebral ligament, which is thickened at the canthi to form the inner and outer tarsal ligaments. The inner tarsal ligament, which can be made to stand out tensely beneath the skin at the inner canthus by drawing the lids outwards with the finger placed over the outer canthus, forms a guide to the situation of swellings in this position; it is attached to the nasal process of the superior maxilla in front of the lachrymal sac. It has also a second connection with the lachrymal bone behind the sac, which it thus embraces. Beyond the sac the ligament terminates in two processes to join the inner ends of the tarsi. The outer tarsal ligament is shorter and less marked.

The free edge of the tarsus forms the long narrow margin of the lid; it is rounded in front where the lashes emerge in rows, and has a sharp edge behind in contact with the globe. The end of this sharp margin is marked, near the inner canthus, by the punctum lachrymali; beyond this the edge of the lid is rounded over the canaliculus, which runs beneath the tarsal ligament to reach the lachrymal sac. On drawing the lids away from the eye the posterior surface of the tarsus is exposed, covered by the thin, smooth, and adherent palpebral conjunctiva, through which is seen the outline of the Meibomian glands, whose orifices open in a row on the free border of the lid near its posterior edge. By pressing the tarsus gently against the globe whilst the eyes are kept open, a row of beads of secretion can be made to issue from the mouths of the glands. This lubricating secretion prevents excoriation when the lids meet together in winking, and also keeps the normal amount of tears from overflowing down the cheek.

Muscles.—Between the palpebral fascia and the skin is the orbicularis palpebrarum, a thin muscle whose fibres surround the palpebral fissure concentrically, and extend from the edge of the lids to beyond the orbital margin, where it joins the contiguous muscles of the face and forehead. The palpebral portion of the muscle, which arises from the internal tarsal ligament, performs the function of a sphincter to the lids in winking, and when the eyes are gently closed, as in sleep; the outer or orbital portion is brought into action during the forcible closing of the lids. The whole muscle, which is supplied by the facial nerve, acts as the depressor of the upper and elevator of the lower lid.

The upper lid is raised by the levator palpebræ; and the lower lid is depressed when the eyes are open and looking straight in front by the relaxation of the orbicularis, which allows the lid to sink by its own weight; but when the

eyes look down, the lower lid is further depressed and drawn backwards by the inferior rectus muscle, which is indirectly connected with the attached margin of the lower tarsus through an expansion of Tenon's capsule.

The levator palpebræ, which arises at the apex of the orbit, and is attached by a thin and wide tendinous expansion to the upper margin and anterior surface of the tarsus, is supplied by the third nerve. It draws the tarsus upwards and backwards over the globe, and forms a fold in the skin corresponding to its line of insertion in the upper tarsus. By raising the outer canthus when looking up the levator also draws up the lower lid slightly. Müller's muscle, which is supplied by the sympathetic nerve, and helps to keep open the lids, consists of unstriped fibres that arise amongst the bundles of the levator palpebræ, and are inserted with its tendon in the tarsus. Similar fibres in the lower lid extend from the inferior rectus to the lower tarsus. The occipito-frontalis, by contracting when the lids are open to their widest extent, can raise the eyebrows, but is unable to affect the position of the tarsus.

Conjunctiva.—The inner surface of the lids is covered by the palpebral conjunctiva, which is thin and smooth over the tarsal, but thicker beyond them where it joins the ocular conjunctiva in the cul-de-sac (*see* "Conjunctiva").

The ophthalmic division of the fifth supplies sensory nerves to the lids.

The arteries of the lids are derived principally from the facial and temporal and also through the lachrymal branch of the ophthalmic. The veins empty into the facial and the ophthalmic.

The lymphatics of the lids pass over the margin of the orbit at the outer canthus to enter the pre-auricular gland.

Congenital Defects

Absence of the lids is as rare a condition as complete adhesion of the lids, and are usually associated with defective developments of the eye. Cysts in the lower lid will be considered in connection with microphthalmos.

Coloboma of the Lids.—Coloboma of the lid is present when the whole thickness of the lid is divided by a vertical fissure which may extend any distance from the edge of one lid up to the orbital margin. It occurs more frequently in the upper lid than in the lower. The fissure may be single or double, and is not infrequently associated with a dermoid growth on the cornea corresponding in its situation to the cleft in the lid. The treatment consists in rawing the margins of the fissure, preserving if possible the tissue at the free edge of the lid to fill up any gap which may be left there when the sides of the coloboma are brought together with sutures; one of these should be passed through the tarsus at its free edge along the lid border.

Dermoid Cysts.—These tumours which most frequently occur near the margin of the orbit have a similar origin to the dermoid growths elsewhere. An anæsthetic is given, and the cyst is opened freely by a single incision, which is made through the skin and cyst wall. The contents are expressed, and the sac is removed completely, principally with the aid of two pair of dissecting forceps. The skin wound is closed with sutures and covered with a dressing.

Epicanthus.—The fold of the skin, which exists over the caruncle at the inner canthus in some Oriental nations, may be so exaggerated in Europeans as to form a congenital defect or deformity. In mild cases, when the bridge of the nose grows more prominent, the defect may disappear entirely or partially, and even in marked cases it may become less pronounced. In bad cases, where the palpebral fissures are small and narrow, and the bridge of the nose is flat and measures from 30 mm. to 40 mm. between the inner canthi, no great improvement need be expected to occur; and even the removal of an elliptical-shaped piece of skin from over the bridge of the nose does not usually improve the patient's appearance.

Other congenital defects—ptosis, distichiasis, symblepharon, and ankyloblepharon—are described subsequently, as their treatment is the same as for the acquired varieties.

Affections of the Skin of the Lids

Erysipelas.—When the lids are involved in an attack of erysipelas, the swelling of the subcutaneous cellular tissue prevents the patient opening the eye until the inflammation is subsiding, a condition which causes him some alarm; it is only, however, when suppuration in the cellular tissue occurs that anxiety need be felt, as the inflammation may lead to sloughing of the tissues of the lid, or extend to the orbit and cause loss of sight through optic neuritis and atrophy, or even to loss of life through purulent meningitis.

The local treatment in the early stages is to apply evaporating lead lotion on lint, which is laid over the face and kept constantly wet. Directly pus can be detected a free incision must be made in the lid, and even then the skin of the lid and also the tarsus may slough away. When this occurs the raw surface must be grafted as soon as possible, and if it is necessary the margins of the lids should be rawed and joined by sutures to prevent, by their temporary union, undue shrinking of the parts. (*See* Lagophthalmos, p. 227.)

Herpes Frontalis. — Herpes of the first division of the fifth nerve begins with neuralgic pains in the region of the affected nerve, and is followed, within a day or two, by the eruption of a crop of herpetic vesicles which appear over the course of one or more of its branches. The affected parts become inflamed and swollen, and often on account of the œdema the lids cannot

be opened for the efficient examination of the cornea and iris; when this occurs vesicles should be looked for upon the side of the nose, over the terminal branch of the nasal nerve (which gives off in the orbit the sensory root to the ciliary ganglion and the long ciliary nerves), to ascertain if it is probable that the cornea, iris, and ciliary body are involved. The vesicles soon begin to dry up and form scabs, which separate and leave depressed, characteristic scars as a permanent evidence of the attack. When the ciliary branches are involved a keratitis or a kerato-irido-cyclitis ensues, which lasts for months, and may end, in spite of all treatment, in the partial or complete destruction of sight. The neuralgia which accompanies an attack usually subsides before the end of two years, but instances occur where tic-douloureux is established, for the relief of which removal of the Gasserian ganglion has been prescribed. The cause of the inflammation of the Gasserian ganglion, the primary seat of the affection, is doubtful.

Treatment.—The indication is to relieve pain, when the skin only is affected. In the inflammatory stage, lint kept constantly wet with evaporating lead lotion is applied over the head and face, and narcotics are given. For the neuralgia, gelsemin, bromides, phenacetin, and allied drugs, as also quinine, iron, cod-liver oil, etc., are prescribed. For the treatment of the affections of the globe produced by herpes, see "Cornea," "Iris and Ciliary Bodies."

Eczema.—When the lids are affected in an attack of eczema, due to some general cause, the treatment does not differ from that for the other parts of the body, except that liquid applications, which would irritate the conjunctiva, must be avoided, and be replaced by emollient ointments such as zinci cremor, acidi borici, etc.

Eczema of the lids which is secondary to some affection of the eyes, such as is produced by the use of atropine or some of its derivatives, can be recognised by its occurrence during the use of the drug, and, usually, by the inflammation being confined to the face; occasionally, however, the dermatitis may become general, and be followed by exfoliation of the whole of the epidermis, including the nails. The treatment consists in stopping the use of the drug and in changing it for another mydriatic, either duboisine or homatropine, and otherwise treating the case as one of general eczema. If at any future time atropine is again used, however long the interval may be, it is likely to produce a similar attack.

Eczema, which is associated with, or secondary to, phlyctenular or catarrhal ophthalmia, and is accompanied by a nasal discharge, is best treated by the application of a 1 per cent solution of nitrate of silver to the conjunctiva, skin, and anterior nares. After the discharge has been removed, the patient being anæsthetised, the silver solution is applied vigorously to the whole of the raw surface, which is then covered with dilute nitrate of mercury ointment (1 part to 4 or 8 of yellow vaseline). The subsequent treatment consists in keeping the parts clean with a solution of borax and anointing them with the ointment.

Abscess.—Abscess in the lid or eyebrow caused by the inflammation of a hair follicle, or associated with suppuration of the cellular tissue of the lid, does not require any special treatment, except that the incision to let out the pus cannot be made too soon, to prevent burrowing in the loose cellular tissue, which may lead to destruction of the delicate skin of the lid. When periostitis of the margin of the orbit is the cause of the inflammation, the abcess cavity must be scraped out with a sharp spoon and disinfected with perchloride of mercury lotion, 1 in 1000, to endeavour to prevent loss of bone and the formation of a sinus, which would lead to the displacement of the lids. (*See* Ectropion, p. 226.)

Furuncle, carbuncle, and malignant pustule do not require special treatment on account of their being situated on the lids.

Ulcers.—Tuberculous ulcer and lupus of the lids are recognised by the same characteristics which these diseases present elsewhere. They frequently extend from the palpebral conjunctiva and destroy a portion of the lid margin before reaching the skin, though they may be secondary to lupus of the lachrymal sac which has spread upwards from the nasal mucous membrane. The enlargement of the pre-auricular gland usually subsides when the disease is cured.

Treatment.—The infiltrated area is scraped with a sharp spoon until the whole of the diseased tissue is removed. The parts are washed with perchloride of mercury, 0·1 per cent, or a chloride of zinc lotion, 4 per cent, and covered with an antiseptic dressing, which is renewed until healing is complete. When the ulcer is finally healed, any displacement of the lid may be remedied by a plastic operation.

Syphilitic and vaccine ulcers of the lids do not require any special treatment on account of their situation.

The differential diagnosis between lupus, hard chancre, and vaccinia of the lids does not give rise to any serious difficulty when an accurate history of the case can be obtained. The slow progress, absence of hardness, and characteristic infiltration around the margin of the ulcer in lupus, render it difficult to confuse the condition with primary chancre of the lid, though the pre-auricular gland may be enlarged in both cases. If the diagnosis is doubtful for a time, the stationary character of the chancre at length followed by secondary symptoms will soon make it clear.

The acute symptoms produced by vaccinia on the lids, its rapid subsidence, and its usual occur-

rence in young mothers, are sufficient to render the diagnosis easy.

Œdema.—Œdema of the lids is usually dependent upon some ascertainable local or general cause; occasionally, more especially in children, it occurs, apparently as a primary affection which disappears quickly without treatment. Œdema due to a distension of the lymphatic vessels of the lids and neighbouring parts may come and go, or remain and resist all treatment. Excision of portions of the hypertrophied subcutaneous tissue, or its destruction with the actual cautery, to allow the lids to be opened, may be tried.

Chromidrosis is a rare condition in which the sweat glands at the lid margin secrete a blue pigment. This appearance can be readily simulated by hysterical patients. (*See* Skin, Disorders of Glands.)

Parasites.—Filaria Loa, a parasitic worm, has been removed from the lids of people who have lived in Old Calabar. The associated clinical features are described under Filariasis (*Filaria loa*).

Affections of the Glands of the Lids

Blepharitis Marginales, or Sycosis Tarsi.—The essential change in sycosis is an inflammation of the follicles and glands of the lashes. In mild cases this causes an excess of secretion from the sebaceous and Meibomian glands, and leads to an exfoliation of the epithelium, which collects in dry scales on the skin between the lashes. When this secretion is removed the lids appear normal except for the congestion of their edges. In severe cases pustules form round the roots of the lashes; these discharge and leave small ulcers, each with a lash in its centre. The discharge collects and forms a crust on the lid margin, which mats the lashes together. When the lash is shed the ulcer heals, but as soon as a new lash appears the same process is repeated. If the disease is not arrested by treatment the follicles gradually atrophy and the lashes eventually cease to grow, after passing through successive stages of degeneration.

During the progress of the disease the skin around the lashes contracts, and the conjunctiva along the lid margin, which is now congested and thickened, becomes everted until it forms the lid border; the lashes and lid border are then found on the outer surface of the lid at some distance from its apparent margin. As the puncta lacrimali are involved in the ectropion, epiphora occurs and keeps up the eversion by producing excoriation and contraction of the skin where the tears flow. In other cases, owing to atrophy of the glands, the lid margins contract, with the result that some of the stunted and distorted lashes are turned in towards the globe and irritate the cornea.

Sycosis may follow any inflammation in which the conjunctiva is involved; often it dates from some febrile attack such as measles.

Treatment.—In all cases the object of the routine treatment is to remove the discharge by washing it away with warm 2 per cent borax lotion, applied with absorbent cotton wool, and when the lids are absolutely clean, some form of mercurial ointment is rubbed into their margins, and this is repeated as often as the discharge collects. The strength of the ointment has to be varied according to the severity of the case; the indication being to employ as strong an ointment as the eye will bear without undue irritation. Ordinarily from half a grain to eight grains of the yellow or of the red oxide of mercury to an ounce of yellow vaseline is prescribed; but in severe cases the dilute nitrate of mercury ointment may be tried.

When the disease is slight the treatment may be entrusted to the patient or to his friends, but when the case is very bad the surgeon should attend to it himself at least once a day. After removing the crust, the lashes in the centre of the pustules and ulcers are pulled out, and the remaining ones are cut off and should be kept short until the healing is completed. The ulcers are then touched with a 1 or 2 per cent solution of nitrate of silver before the ointment is rubbed into the lid margin. A more rapid improvement will take place if the home attendant removes the crusts as often as they collect, and then applies the ointment.

When the conjunctiva on the lid margin is thickened sufficiently to produce eversion of the puncta, it will be necessary to cauterise it on the ocular side of the punctum with the galvanic cautery. The contraction produced by the scar will suffice to reinvert the puncta. Until this is effected the cure will never be complete. If the ectropion is more pronounced it will be necessary to apply Snellen's sutures to restore the border of the lid to its normal position. (*See* Ectropion, p. 226.)

When the disease has caused many of the stunted lashes to be inverted, some form of entropion operation will have to be performed, either a Burow, if the lid is thin, or a Jaesche Arlt, if it is thickened. (*See* Entropion, p. 225.)

In all cases constant attention will either diminish or cure the affection, but if this care is not continued for some time after the cure appears to be completed a relapse is likely to occur; also if the glasses, which correct the error of refraction that almost invariably is present, are not worn, either constantly or at least during near work, the sycosis will recur.

Hordeolum, or Stye.—An acute suppurative inflammation of the sebaceous glands in connection with the roots of the lashes or of a Meibomian gland constitutes, respectively, an external and an internal stye. They may occur after a general febrile attack, such as measles or chicken-pox, or after a muco-purulent ophthalmia or other conjunctival affection, or without any obvious cause. The heat and discomfort

which accompanies the onset of the attack is followed by redness and swelling of the lid, which causes considerable pain until suppuration and the bursting of the little abscess affords relief. A stye which points on the outer or on the inner surface of the lid according to its seat of origin is accompanied by more or less chemosis of the conjunctiva, as it is situated nearer to, or farther from, the outer canthus.

Treatment.—Abortive measures may be tried in the earliest stage before suppuration has begun: this consists in the internal administration of five drops of the tincture of pulsatilla, every ten minutes, for an hour or longer; or of half to one grain pills of sulphurated lime, which are taken every hour, for six hours, or until the attack begins to subside. If this treatment fails or was not begun sufficiently early, then epilation of the affected lash or massage with a glass rod of the implicated Meibomian gland should be tried, to evacuate any retained secretion. When suppuration has begun the pus should be let out at once by puncturing the abscess with the point of a Graefe's knife, which affords immediate relief. Until this can be done hot boric acid fomentations should be used frequently, and should be continued after the pus has escaped until the inflammation has subsided. After an attack the lids must be bathed night and morning with a 2 per cent solution of boric acid, or with a 1 in 4000 solution of perchloride of mercury and then massaged with boric acid ointment to keep the ducts of the glands in a normal condition. The general health should also be attended to, and fresh air and regular exercise be ordered.

In every case the refraction should be examined and corrected with glasses, which are to be worn constantly, at least until all tendency to a relapse has passed.

Chalazion.—This affection of the Meibomian glands, known also as a Meibomian cyst or tumour, begins either as a small painless swelling which may remain unnoticed until it has attained a considerable size, or it occurs after an inflammatory attack on the lid. As the tumour increases in size, by the growth of its granulation-like tissue which is encapsuled in a layer of connective tissue, the swelling becomes more prominent and can be felt in the tarsus beneath the skin. After a time the substance of the growth breaks down in the centre, the liquid contents escape through a perforation in the palpebral conjunctiva, and are followed by a button of the granulation tissue which is flattened by coming in contact with the globe. In this condition the tumour may remain for an indefinite time. When a cyst occurs near the orifice of the gland it usually perforates the skin externally. Occasionally these tumours occur singly, but frequently they appear in crops, and may thicken nearly the whole tarsus. As there is no tendency to a spontaneous cure the lid should be everted after the instillation of a few drops of a 2 per cent cocaine solution, and a little powdered cocaine is dusted over the affected gland, which is easily recognised by its colour. A vertical incision is made into the cyst with a Beer's knife, and the contents are squeezed out. The interior of the cyst is then scraped out with a spoon to remove all the jelly-like material, which grows again if the sac is not completely emptied. After the operation the cavity fills with blood, and the tumour only disappears when this has absorbed. When the sac has perforated spontaneously, the button of granulation tissue should be cut off and the contents scraped out through the opening in the tarsus which may be enlarged with a knife. Occasionally on incising the cyst it is found to consist mainly of connective tissue with very little jelly-like contents. In such cases the growth has to be dissected out through an incision made in the skin. The after-treatment consists in fomenting the lid with boric acid or sublimate lotion, to be followed by a massage of the lids, for a few minutes, every night.

Infarcts of the Meibomian Glands.—When the contents of these glands are retained and become inspissated, they may undergo calcareous degeneration (lythiasis), and during the process of their elimination they make their way through the conjunctiva and act as foreign bodies which irritate the globe.

The concretion should be removed with the point of a needle after the application of a little cocaine solution. Subsequently the patient should massage the tarsus by rubbing boric acid ointment into the edges of the lids every night to keep the glands empty.

Tarsitis.—The tarsus occasionally is the subject of an acute inflammatory attack followed by suppuration and sloughing of the whole of its structure which leads to a total loss of the lashes and to a lax condition of the lid. Even an early incision through the skin and capsule of the tarsus, followed by hot boric acid fomentations to the lids, does not always prevent the progress of the inflammation. A more chronic inflammation of the tarsus which causes considerable thickening of the lid and takes many weeks to run its course is due to tertiary syphilis.

Affections of the Muscles

Spasm of the Orbicularis.—Spasm of the orbicularis varies in degree from an occasional twitching of a few of its fibres (live blood) to the complete closure of the palpebral fissure by the contraction of both the palpebral and the orbital portions of the muscle.

It occurs occasionally as a primary affection, but more usually it is a secondary symptom of an irritation of a branch of one of the divisions of the fifth nerve.

The most common cause of the spasm is irritation of the conjunctiva or cornea by foreign

bodies, inverted lashes, or any of the conjunctival or corneal affections, and also errors of refraction. If the eyes appear to be normal, changes in the other parts supplied by the second and third divisions of the fifth nerve, more especially in the nose, mouth, or teeth, should be treated.

When the cause is removed the spasm subsides without further trouble, unless in its turn it has produced ectropion, or entropion with blepharophimosis, of the lids, to which reference again will be made.

Primary spasm, especially when it is limited to one eye, in young female patients, is usually due to hysteria. In older people the cause may not be discoverable.

The hysterical cases, when both eyes are affected, may often be cured at once by the instillation of a few drops of a 2 per cent cocaine solution, provided the patient is assured that they will have this effect; but when one eye only is attacked the condition may continue for years, if too much attention is paid to it, though eventually complete recovery will occur.

Senile blepharospasm, for which a remediable cause can be found in the eye, ear, nose, mouth, or throat, is likely to recover, but when nothing can be discovered and cocaine and astringent lotions and treatment (tonics and bromides, etc.) for the general health fail, then a canthoplastic operation can be performed or a portion of the orbicularis muscle near the outer canthus can be excised.

Paralysis of the Orbicularis.—Paralysis of the orbicularis, though usually due to an affection of the trunk of the facial nerve, may be caused by some central lesion. The condition leads to epiphora, and sometimes to ulceration of the cornea from exposure of the eye during sleep.

Until the treatment which is directed to the relief of the primary cause, rheumatism, syphilis, injuries, inflammation due to ear disease, growths, etc., has succeeded or is given up as hopeless, the orbicularis as well as the other facial muscles should be galvanised regularly, and during sleep the eye should be protected by closing the lids with a pad and bandage or with strapping. When the lower lid droops and becomes everted, the condition should be remedied by the operation of tarsoraphy, if the paralysis cannot be cured. At the same time the punctum may be reinverted by cauterising the conjunctiva just within the tear point.

LEVATOR PALPEBRÆ — *Congenital Ptosis.* — Congenital ptosis or drooping of the upper lid is usually symmetrical, and not infrequently it is accompanied by an inability to turn the eyes upwards above the horizon even when the lids are held up. The condition is due to the complete or partial absence or to paralysis of the elevator of the lids, consequently the patient wrinkles his forehead and throws back his head in his effort to raise the lids and to look beneath them.

Operative treatment alone affords the patient relief. The choice of operations are numerous, but one of the simplest and most effectual in the majority of cases is the insertion of a wire suture to connect the occipito-frontalis with the tarsus. This also is the object of most of the other ptosis operations.

This operation, which was devised by Dr. Mules, can be practised in the following manner: The patient being anæsthetised, a horn spatule is placed beneath the upper lid, which is incised with a Beer's knife, along the line that divides the lashes from the orifices of the Meibomian glands. This incision extends along the middle third of the lid and half-way through the width of the tarsus. Two straight needles, two inches long, are passed from either extremity of the bottom of the wound, through the tarsus, beneath the orbicularis and over the margin of the orbit until they emerge, about two inches apart, through the lower part of the occipito-frontalis and the skin above the eyebrows. When both needles are in position the palpebral conjunctival should be inspected to see that it has not been perforated. A Beer's knife is now passed along the needles to enlarge the skin wounds at their point of emergence. The ends of a piece of freshly annealed silver wire is passed through the eyes of the needles and drawn through the lid, leaving a loop buried in the bottom of the tarsal incision; the ends of the wire are drawn upon until the lid is sufficiently elevated, and then cut off about an inch from the wounds. Each of these free ends is twisted into a coil, and is pushed through the skin opening into the subcutaneous tissue, and a dressing is applied. The coiled ends of the wire act as knots and can be drawn upon afterwards to lengthen the loop of wire, in case too great an effect was produced at the time of the operation. If at any future time the wire should break, the operation can be repeated.

A congenital condition in which the lid usually droops, but is raised spasmodically when the corresponding internal pterygoid muscle contracts, is a defect which cannot be remedied.

Paralysis of Müller's muscle due to an affection of the sympathetic nerve leads to narrowing of the palpebral fissure, but not to true ptosis. The treatment should be directed to the relief of the primary cause of the paralysis.

ACQUIRED PTOSIS.—Ptosis when acquired is caused by a paresis, or paralysis, of the levator palpebræ due to an affection of the nucleus or the trunk of the third nerve in the cranium or orbit. Tabes dorsalis, gummata, inflammation, tumours, and injuries may be the cause of the affection, and until it is relieved by internal treatment nothing further need be done. Even when internal treatment fails, it is rarely necessary to perform an operation to raise the lid, except when both eyes are affected (ophthalmoplegia externa), as this would cause the patient

to have diplopia if the other ocular muscles supplied by the third nerve were also paralysed. In complete double ptosis Mule's operation may be performed on one eye to enable the patient to see better to move about.

Defects in Position and Connection of the Lid

Trichiasis.—Trichiasis, or the growth backwards of one or all of the lashes until they come in contact with the eye, is due to some cicatricial distortion of the tarsus caused by granular ophthalmia, sycosis tarsi, burns or injuries, etc. As a consequence the globe is perpetually irritated, and the patient always has a feeling of having a foreign body in the eye. Until the primary cause has been cured the ingrowing lashes should be pulled out with a pair of forceps as often as they appear. When only a few lashes are misplaced, and the lid margin is normally situated, a permanent cure can be effected by destroying the roots of the faulty lashes with electrolysis. When a part or the whole of the edge of the lid is wrongly directed, then some operation should be performed to restore the lid margin with the lashes to a normal position.

In people under twenty-five years of age a Burow's operation is generally successful, but in all other cases a Jaesche-Arlt operation should be performed.

Burow's operation consists, when the patient is anæsthetised, in reverting the lid over a broad ivory spatula, and cutting with a Beer's knife through the whole thickness of the tarsus down to the orbicularis along a line parallel to and at a distance of 3 mm. from the edge of the lid. The broad furrow thus formed between the two portions of the tarsus is filled up with a mass of connective tissue which allows the narrow strip bearing the lashes to resume its normal direction. In older people this exudation into the sulcus is generally insufficient, and the eversion of the lashes fails to take place. In such cases the following slightly modified *Jaesche-Arlt operation* is indicated :—

The patient is anæsthetised, and the lid is clamped between the blades of Desmarre's forceps, care being exercised to evert the lid margin as much as possible to facilitate the next step. This consists in making an incision with a Beer's knife from one extremity to the other of the tarsus, along the line on its free border, which separates the orifices of the Meibomian glands from the lashes, thus dividing the tarsus for two-thirds or more of its depth into two laminæ. The raw surface of the posterior flap is dried and carefully inspected for the roots of any lashes which may have been cut through ; they appear as small black bulbous points which are dissected out. A strip of skin and muscle about 3 mm. wide is excised by two parallel incisions which extend from end to end of the lid and meet together at their extremities. The removal of this strip of skin and muscle along a line about 1·5 mm. above the lashes should expose a corresponding portion of the tarsus. The margins of the skin wound are united by three sutures, the forceps are removed, and the lids are covered with a dressing. In a few days the wound is healed, and the lashes are found to be in a normal position. Even when only a portion of the lashes is inverted it is better to treat the whole lid in this manner.

Distichiasis.—This congenital abnormality consists in the growth of a second row of fine delicate hairs from the posterior margin of the free edge of the lid behind the normal row of lashes.

If the abnormal lashes are left alone, and are not pulled out and made coarse, they give rise to no inconvenience.

Entropion. — Entropion exists when the margin of the lid with the lashes is inverted until the skin comes in contact with the globe. It is due either to a contraction of the orbicularis muscle, spastic entropion, or to scars in the conjunctiva or tarsus, cicatricial entropion.

Spastic entropion is seen only in old people with a lax and superabundant amount of skin, which allows the lid margin to turn in when it is not well supported, because the globe is shrunken or absent, or because of blepharospasm. This form of entropion is limited practically to the lower lid. Cicatricial entropion varies only in degree from trichiasis ; it is due to similar causes, and is treated in the same way.

The treatment of spastic entropion consists in removing any cause which may have produced it, such as the prolonged wearing of a cataract bandage, which should be exchanged for dark glasses or a shade. If no cause can be found the lid should be kept everted with strapping, and if, after a good trial, this fails, then Galliard sutures should be inserted, or even a piece of the redundant skin and muscles may be excised.

Galliard's suture is applied in the following manner : A large curved needle threaded with No. 2 plaited silk suture is entered in the middle of the lower lid immediately below the lashes, and is passed beneath the orbicularis muscle and made to emerge through the skin on a level with the orbital margin. The ends of the suture are then tied together as tightly as possible over the fold of skin. In bad cases two similar sutures may be applied on either side of the central one, midway between it and the ends of the lid. They are not removed until they have cut their way almost out through the skin.

If this fails a strip of skin and orbicularis muscle is excised with scissors, and the wound is closed with sutures. The width of the strip is ascertained in each case by picking up with the T-shaped forceps a fold of skin in contact with the lid margin ; it should be just of sufficient breadth to restore the lid to its

normal position. Care, however, must be taken to remove all the exposed orbicularis muscle. The wound is closed with fine sutures, and covered with a pad and bandage until it has healed.

Ectropion, or eversion of the lid, is produced by the following causes:—Spasm of the orbicularis muscle, paralysis of the orbicularis muscle, senile changes in the lids and cicatrices.

Spastic ectropion, which may affect both lids, occurs in the young who are suffering from some conjunctival inflammation which produces blepharospasm. It disappears when the primary cause is cured, and therefore requires no special treatment.

When paralysis of the orbicularis muscle is the cause of the drooping of the lower lid the treatment will depend on the length of time it has existed, and upon the nature of the primary cause. If the paralysis is likely to be cured it will be necessary only to support the lid with strapping or with a pad and bandage until this has been effected, but when the paralysis is permanent the lax and elongated lid can be shortened and restored almost to its normal position by excising a wedge-shaped piece from its extremity at the outer canthus. This is done by making an incision from the outer canthus through the whole thickness of the lid outwards and downwards for about an inch. The lower lid is then drawn outwards until its margin is in contact with the globe, and the portion which now extends beyond the canthus is removed by an incision which joins the end of the first cut. Sutures are inserted to close the wound, special care being taken to bring the free margin of the lids together at the outer canthus.

Senile ectropion, which is chiefly due to a loss of elasticity and tone in the tissue of the lid, may begin by an epiphora due to the eversion of the punctum started by a conjunctival catarrh. When once the tears flow over the cheek a vicious circle is established which may lead to the complete eversion of the whole of the lower lid, together with a thickening of the palpebral conjunctiva, caused by the irritation of the lashes of the upper lid.

In the earliest stage, when only the punctum and inner end of the lid are everted, they may be restored to their normal position by means of a scar, produced by burning the conjunctiva on its inside with the actual cautery. At the same time the skin, where the tears flow, should be kept supple with ointment.

When the whole length of the lid is everted, the effect should be tried of Snellen's sutures, which are applied in the following manner:—

The patient may be anæsthetised, or cocaine is applied and injected beneath the lid. A suture, with a two-inch straight needle at each end, is taken, and one of the needles is passed from the highest point of the everted conjunctiva, a little to one side of the middle of the lid, beneath the orbicularis and out through the skin just below the lower margin of the orbit. The other needle is passed in a similar manner, and parallel to the first, at a point at about one centimetre to its outer side. The loop formed by the suture on the top of the conjunctiva is now drawn tight, by tying the ends together over a piece of rubber tubing until the margin of the lid is inverted. Two similar sutures are usually inserted on either side of the central one before it is tied. The sutures are removed as soon as they begin to cut their way out.

Cicatricial ectropion is due not only to loss of tissue caused by burns, injuries, ulcers, and abscesses, but also to sycosis and to eczema of the lids associated with conjunctival and lachrymal affections. The latter can be dealt with by cauterising the everted conjunctiva with the actual cautery, or by Snellen's sutures.

When the ectropion is caused by a scar in the skin adhering to the bony margin of the orbit, the adhesion should be divided subcutaneously. The patient is anæsthetised, and a strong Graefe's knife is entered at a point about an inch distant from the scar, which is freely separated from the bone without buttonholing the skin. The knife is withdrawn, and the cavity thus formed fills at once with blood, which raises the scar to a level with the surrounding skin. This level is maintained for some days by passing two hare-lip pins at right angles to each other through the normal skin and across the cavity. If necessary the margins of the lids may be brought together with strapping, or united temporarily with sutures, until healing has taken place.

When the loss of tissue is not great and the lower lid alone is drawn down, the defect can be remedied by Warton Jones' V Y operations. The patient is anæsthetised, and an incision is made through the skin and subcutaneous tissue from just below the inner canthus, obliquely down and out to a point below the lowest part of the scar. A second incision from near the outer canthus is made to join the end of the first. The V-shaped flap, which should not be too pointed, is dissected up until the margin of the lid can be raised to its normal position, where it is retained by one or more hare-lip pins. One of these pins is passed from one side to the other of the wounds, transfixing and securing the flap in its new position, where it is retained by a figure-of-eight suture made over its ends. The leg and sides of the Y are united by interrupted sutures. When union has taken place the hare-lip pin is removed, and the edge of the lid remains at its normal position.

If the loss of tissue is too great to be remedied in this manner, an incision can be made in the skin or scar of the lower lid parallel to its free margin. If the raw area is not too large it can be covered by a flap of skin with a pedicle, which is dissected up from the neighbouring

parts, turned round and stitched into its new place. Or a flap of thin skin may be detached from a distant part of the body and stitched into position. As such detached flaps always shrink, both when they are dissected out and also when they have taken root, it will be necessary when marking out its size and shape to allow at least one-third for shrinkage. Or, in very severe cases, after preparing the area the edges of the lids are united with sutures, and the raw surface is then allowed to granulate over until it is ready to be covered with skin grafts. Owing to the subsequent shrinkage which takes place in all these cases it will probably be necessary to repeat the operation at a later date.

Ankyloblepharon.—Ankyloblepharon, which consists in the union of the margins of the lids only, is usually associated with symblepharon, where the lids are adherent to the globe.

Both conditions are due to the healing together of raw surfaces produced by some injury or disease, such as burns, diphtheritic ophthalmia, pemphigus of the conjunctiva, etc. When the margins of the lids are joined together, without any adhesion being present between the lids and the globe, their separation is effected by dividing the scar of their union with scissors, and keeping the raw surfaces apart by everting the lower lid by strapping. When the adhesion involves the canthus, it will be necessary to unite a piece of conjunctiva to the skin at the angle of the lids to prevent their readhesion.

Symblepharon.—Symblepharon is the cicatricial adhesion of the lids to the globe, produced by the partial or complete destruction of the conjunctiva. It occurs after burns, injuries, and such diseases as diphtheria and pemphigus, which denude the opposing surfaces of the ocular and palpebral conjunctiva. When the adhesion does not include the fornix, so that a probe can be passed beneath it, the condition is called anterior symblepharon, and can be remedied by dividing the adhesion and stitching together the contiguous edges of the ocular conjunctiva, leaving the raw surface on the lid to granulate.

Posterior symblepharon implies the destruction of the fornix, and may be partial or complete; in the latter case the conjunctival sac is obliterated, and, as the lids are adherent to the cornea, the vision is practically destroyed.

The treatment of cases of partial symblepharon is far from satisfactory in the majority of instances, and therefore, if the condition does not give rise to urgent symptoms, it is better to wait until the scar has had time to become soft and elastic, when, often, it is found that all discomfort has subsided.

The reason of the failure to keep the lids apart in complete symblepharon is that healing takes place from the bottom of the cul-de-sac, and pushes up any grafts or portions of the conjunctiva or skin which have been transplanted there.

Blepharophimosis.—Blepharophimosis, which consists of an apparent shortening of the length of the palpebral fissure, produced by a fold of skin being dragged in front of the outer canthus, is associated with, and is directly produced by, affections of the conjunctiva, which give rise to blepharospasm.

The condition subsides when the primary cause disappears, or it can be cured in the same way as blepharospasm by the operation of canthoplasty, which is performed in the following manner. The patient being anæsthetised, the outer ends of the lids are put on the stretch and held slightly apart, while the palpebral fissure is enlarged by dividing the tissues at the outer canthus with a pair of blunt-pointed scissors, one blade of which is passed into the conjunctival sac. If the contraction of the fissure is very marked the palpebral ligament should be divided vertically up to the orbital margin by cutting it through with the scissors passed into the wound, where the sharp edge of the fascia can be felt, between the skin and conjunctiva, when the upper lid is put upon the stretch. If it is intended to produce a permanent enlargement of the fissure, the conjunctiva and skin are united together at the outer angle of the wound by a suture, and two other sutures are inserted similarly in the upper and lower parts of the wound. If only a temporary effect is required the sutures are not inserted.

Lagophthalmos.—Lagophthalmos exists when the lids do not meet together during sleep. This may be due to a congenital or pathological shortness of the lids, to ectropion, to a paresis, or paralysis of the orbicularis, or to the globe being too large or too prominent for the lids to close over it. The effect this condition produces will depend upon the degree to which the eye is exposed. When the chink is narrow it may only cause a slight chronic conjunctival irritation, but when a part or the whole of the cornea is exposed, the latter may inflame and ulcerate, or become opaque, and lead to partial or to complete loss of sight.

When the lids are too short congenitally there is no remedy beyond the regular use of a borax or boric acid lotion to keep the conjunctival sac clean. If the condition is due to, or is associated with, sycosis or eczema, then the treatment for these affections should be carried out; and when it is due to ectropion an appropriate operation should be performed.

The exposure of the eye which occurs during prolonged unconsciousness, in any illness, is to be remedied by bathing the eyes with boric acid lotion and keeping the lids closed with a narrow piece of strapping.

For paralytic lagophthalmos a protecting

bandage or the strapping of the lids together at night is indicated until treatment for the primary cause succeeds. When the orbicularis does not recover from the paralysis the lagophthalmos may be lessened, if an operation for ectropion of the lower lid has to be performed, but can never be cured, and the eye will always have to be protected by means of glasses in the daytime, and by the closure of the lids at night.

When the lids cannot be made to meet over a prominent or enlarged eye, except when the palpebral fissure is shortened by bringing the lids together at the outer canthus, then the operation of a tarsoraphy should be performed; if this does not suffice, the margins of the lids should be sewn together throughout their whole length except for a narrow chink in the centre.

Tarsoraphy.—Previous to anæsthetising the patient or to injecting cocaine subcutaneously near the outer canthus the lids are pinched together at the external canthus, to ascertain the extent to which they must be united; this point is marked on the skin. If it is likely that the proptosis, which is usually due to Graves' disease, will subside, a thin slice is taken away from the margin of both lids up to, but not including, the angle of the outer canthus, which will allow of the fissure being reopened subsequently. If the condition is not likely to improve, then a thicker piece of tissue, including the canthus, is removed with a Beer's knife. The raw surfaces on the lids are united with sutures, and the remaining portion of the lids is brought together with strapping until union is complete.

Growths on the Lids

Benign. — *Xanthelasma.* — Patches of xanthelasma generally occur on the lids below the upper and inner angle of the orbit. Though not infrequently they are associated with bilious headaches, no treatment but excision will effect their removal.

Excision of the growth, at the patient's wish, should only be carried out when its removal will not produce ectropion of the punctum, which would occur if a large piece of the skin had to be taken away.

Molluscum contagiosum.—When these tumours are not very numerous they can be removed by expression, but if they are scattered over the whole of the body, only the large ones can be treated in this manner, sulphur ointment being rubbed into the remainder, or sulphur baths are prescribed. An anæsthetic is given when the tumours to be removed are numerous or inflamed, and then, with a pair of Graddy's forceps, a fold of the skin containing the tumour is gripped between the forcep blades and the contents are squeezed out. Before doing this the skin over the apex of the growth may be incised if the tumour is very large or is inflamed and suppurating; in the latter case it may be necessary to scrape out the growth with a sharp spoon. The wounds in the skin are dressed with boric acid powder and shreds of absorbent cotton-wool.

Milium and Cysts. — Milia, which appear frequently in the skin of the face below the lids, can be removed by expressing their contents through an opening made into their cavity with the point of a sharp needle.

The clear cysts which occur on the lid border by the occlusion of the glands of Moll (Fuchs) are opened freely by transfixion with the point of a Graefe's knife. Powdered cocaine is then dusted into their interior before the lining membrane is removed, wholly or in part, by fine forceps and scissors.

Nœvus. — Nævoid growths in the lids may involve the skin, the subcutaneous tissues, or the palpebral conjunctiva, and then extend deeply into the orbit. The patients are usually brought when quite young, either for the relief of the disfigurement or because the tumour is growing. The treatment consists in destroying the vascular tissue with the galvano-cautery, or by electrolysis (*see* "Capillaries" and "Electrolysis"). When the skin only is involved its surface is burned by touching it in many places with a fine, red-hot cautery point. This is repeated at intervals until the skin has lost the nævoid colour. When both the skin and deeper structures are involved, the cautery point is passed through the skin into the tissue beneath, where it is made to burn a small cavity. This is repeated at several points and usually on several occasions. When the deep tissues only are involved, two or more platinum needles connected with the pole of a constant current battery are plunged deeply into the affected part, and the current is slowly applied until gas bubbles begin to appear in the wounds. After half a minute the current is reversed. At the first sitting it is not well to make more than two or three punctures until the effect produced by the treatment has been watched. In this way the blood is coagulated in the vessels, and the tissue shrinks without producing any marked scarring of the skin. The wounds are kept aseptic by careful attention to the dressings. An anæsthetic is required at each of the sittings, which may have to be repeated several times in the case of large growths. If the conjunctiva alone is involved, cocaine will suffice as an anæsthetic when the growth is destroyed by the galvano-cautery.

Moles.—Black moles, or those which are only pigmented, should be excised, more especially if they occur on or near the margin of the lid. Warts and horny growths on the lids should be removed after a few drops of cocaine solution have been injected into the subcutaneous tissue beneath them, or if seated on the lid margin, after powdered cocaine has been dusted over them. The wound in the skin is closed by a suture, and covered by a dry dressing.

Fibromata, or simple mollusca, are removed in the same way.

Malignant.—*Rodent Ulcer.*—The skin of the lids, more especially near the inner canthus, is frequently the seat of a rodent ulcer, which may exist as a small raised nodule of infiltration with a central area, covered with a scaly crust, which ulcerates and heals without spreading to any perceptible degree for months or years; the small surrounding nodules contain new vessels on their surface. As long as the ulcer is confined to the dry skin it grows very slowly, but when once it has invaded the mucous membrane on the lid margin it spreads fairly rapidly. The pre-auricular gland is not enlarged.

The treatment consists in removing the growth together with a broad surrounding margin of healthy tissue. When this is done it has no tendency to recur, provided the skin only is involved; but when the mucous membrane is attacked even a very free removal will not always prevent a relapse. When so much of the lids has to be removed that the globe is left exposed and cannot be covered by a flap from the neighbouring parts it is better to perform exenteration of the orbit. When the bony walls are invaded they must be freely removed.

Epithelioma

Growths of this nature on the lids are rare. They may begin as a small wart on the free edge of the lid, which increases slowly in size until eventually its centre breaks down and ulcerates. By a continuation of this process the lids and surrounding tissues are destroyed and the neighbouring lymphatic glands (pre-auricular and submaxillary) are affected.

The disease may be simulated by broken-down sebaceous cysts, molluscum contagiosum, and fungating Meibomian cysts, but an exploratory incision or a microscopic examination will determine the diagnosis.

The treatment consists in the free and complete removal of the growth and the enlarged glands.

Sarcoma

Sarcoma involving the lids may occur at any age. In infancy it usually begins in the orbit and involves the lids secondarily, and is of the round or spindle-shaped cell variety, whereas in adults it begins generally in some pigmented mole. If the diagnosis is doubtful an exploratory incision should be made and a portion of the growth examined microscopically. In either case complete removal of the growth is essential, and if necessary the whole of the lids and orbital contents must be sacrificed.

Restoration of an Eyelid.—Whenever the lid with the palpebral conjunctiva has been removed, an attempt should be made to remedy the loss by means of a pedunculated flap which may be formed from the skin of the forehead, temple, or cheek.

In the case of growths it is better to wait until healing of the primary operation wound has taken place before performing any plastic operation, as by judicious drawing of the neighbouring skin and ocular conjunctiva together even the removal of extensive portions of the lids may be recovered from to an unexpected degree.

Injuries to the Lids

Ecchymosis of the lids, however great the swelling may be, subsides without suppuration under the application of evaporating lotions, provided the skin is not torn, the orbital bones are not broken, and the eye is not injured. When the skin of the lid is wounded the progress of the case will depend upon the cause of the injury. Simple incised wounds, and contused wounds caused by a blow with the fist against the margin of the orbit, which may have the appearance of being incised and lead to an error of diagnosis, should be washed and rendered aseptic and then closed with sutures and covered with an antiseptic dressing. When the edge of the lid has been cut through, a silk suture should be passed through the tarsus at its free border and the skin wound carefully united, otherwise a notch is likely to be left in the palpebral margin which would cause epiphora and considerable disfigurement. When this has occurred the lid should be divided along the original scar and united. This is called for especially when the lid has been torn through at or near the inner canthus and across the canaliculi.

In such cases the nasal portion of the canaliculus should be divided and kept open to prevent its permanent closing.

Surgical emphysema of the lids indicates that the injury has broken some of the orbital bones which are covered by the mucous membrane in connection with the air-passages. Through the rent in the membrane air is forced into the subcutaneous cellular tissue during the effort which the patient makes to get rid of the blood by blowing the nose. The treatment is to avoid blowing the nose and to keep the parts covered with a spirit or evaporating lead lotion until the swelling has subsided.

Eyesight. *See* Eye, *and cross-references*; School Children, Examination; Vision; etc.

Eyestrain.—Eyestrain has of late years been much dwelt upon as a cause of the prolonged ill-health which many well-known scientists and literary men have suffered from. *See* Asthenopia; etc.

Face. *See* Abdomen, Clinical Examination (*Appearance of Face*); Abdomen, Injuries of (*Traumatic Peritonitis, Face*); Arteries, Ligature (*Facial*); Ascites (*Tuberculous Peritonitis, Appearance of Face*); Asthma (*Symptoms*);

Atrophy, Infantile (*Symptoms*, *Face*); Brain, Tumours of (*Localising Symptoms in Face*); Eczema (*Regional Forms*, *Face*); Facial Hemiatrophy; Facial Hemihypertrophy; Facial Nerve, Paralysis of; Facial Spasm; Heart, Myocardium and Endocardium (*Symptomatology*, *Dyspnœa*, etc.); Labour, Diagnosis and Mechanism (*Face Presentations*); Myasthenia Gravis (*Symptomatology*); Physiognomy and Expression; Tetany (*Symptoms*, *Motor*, *in Face*); Teratology (*Malformations of Face*); Toxicology (*Strychnine*, *Symptoms*, *Face*).

Facial Hemiatrophy.

As the name implies, this affection is characterised by atrophy of one-half of the face, a defect which is shared by the skin, subcutaneous tissues, and bone; but all regions of the face do not suffer in equal degree.

Etiology.—A direct hereditary transmission of the affection has been traced in a few cases. The manifestations of the malady most commonly appear before puberty, and no genuine case of the kind has been known to commence after the age of thirty years. Of the recorded cases, about a hundred in all, more females have been attacked than males. Among the possible exciting causes of the affection trauma demands special attention, as an injury to the affected side of the face has preceded the manifestations of the facial hemiatrophy sufficiently commonly to suggest a causal relationship. Other conditions that have been regarded as similarly operative are abscess in the region of the ear, tonsillitis, erysipelas, influenza, and various of the specific fevers, such as scarlet fever, measles, and enteric fever; but in a considerable number of the cases no cause has been ascertained.

Symptoms.—A white or yellowish white patch on the cheek, chin, or forehead is, as a rule, the earliest indication of the malady. There may be more than one such patch, in which case, as each spreads, they tend to become confluent, and a large area of the skin of the face is thus involved; but in any case there is a gradual spread of the process from the seat where it first appears. The margins of the patch are not well defined at first, but they become more so in time. The skin may or may not be glossy, but in any case it is parchment-like, and the part has a hidebound appearance; the cutaneous excretions, including sweat, may be diminished on the affected side, and when parts that are covered by hair are involved, as for instance the eyebrows, the hairs fall out or become changed in colour.

As the morbid process develops further there is atrophy of the subcutaneous tissues, and there results a local depression which is most obvious when the seat of the change happens to be the cheek, as is most commonly the case, a deep pit appearing beneath the malar bone. So too in consequence of the loss of the orbital fat the eyeball sinks in on the affected side. In the further progress of the disease not only does a wider and wider area of skin, including subcutaneous tissues, become involved, but the muscles share in the wasting, so that in cases of long standing their volume is much diminished, not, however, owing to any real atrophy or degenerative change of the muscle fibres, but merely in consequence of disappearance of the interstitial fat. That there is no change in the muscle fibre proper is proved clinically by the fact that the facial movements are unaffected, except in so far as they are hampered by the hidebound condition of the skin; and, moreover, on electrical examination they are found to respond normally, except that it frequently happens that on faradic excitation a response is obtained from the muscles with a weaker current than that required to evoke contraction of the muscles of the unaffected side, in that the current meets with less resistance in its passage to the muscles in consequence of the atrophy of the subcutaneous tissues.

The bones of the face also share in the atrophy, so that the malar, frontal, and superior and inferior maxillary bones may in time all become much diminished in size, notably, though not exclusively, in the cases in which the disease becomes manifest at or before the period of puberty, and the teeth have been found smaller and decayed on the affected side in the child. The cartilaginous tissues are also affected as a rule, so that the cartilages of the nose may become shrunken, as may those of the ear. The rate of progress of the disease varies in different cases, and there is also a considerable difference in its extent, for in some patients it is limited to the part in which it first appears, most commonly the cheek, while in others the whole of the structures on one-half of the face become involved. The change is absolutely limited by the middle line; but in consequence of the shrinking of the affected half of the face parts of the healthy side encroach on that which is atrophied; notably is this the case in regard to the chin and nose.

The term hemiatrophy is not applicable to all cases of facial atrophy, in that similar changes have been met with on both sides; so too the face may not be alone affected, for the tongue may share in the unilateral diminution of bulk, a state of things that, as in the case of the facial muscles, does not depend on a true atrophy of the muscle fibres of the tongue, for they respond normally on electrical excitation. Then, again, other parts of the body have been known to be affected, areas of atrophy being found on the back and arm on the same side as the facial affection.

Cutaneous sensibility is preserved over the affected side of the face, and the sense of taste is preserved even in the cases in which the corresponding half of the tongue is diminished in size.

Morbid Anatomy and Pathology.—Five cases have come to necropsy, but in only one of these (Mendel) have the morbid changes been fully described. The facial nerve was intact, but there was proliferating interstitial neuritis of the fifth cranial nerve and atrophy of its descending root, and of the cells of the substantia ferruginea pontis. Microscopical examination of the tissues of the face has revealed atrophy of the papillæ of the skin, with no definite change in the epidermis, and wasting of the subcutaneous fat and connective tissue.

The malady was variously regarded by older writers as a "trophoneurosis," a "vaso-motor change," and a "sympathetic affection," all of which views were purely hypothetical. Another hypothesis is that the condition depends on an arrest of development, though it is not known how this is induced, except that Hutchinson has suggested that it is due to morphœa of the fifth nerve. The view that has gained widest acceptance is that advanced by Mendel, who regards the facial atrophy as the result of an interstitial neuritis of the fifth nerve, the condition present in the case examined by him. Objections have, however, been raised to this view, so that it cannot at present be said that there is any consensus of opinion as to the pathology of the affection; on the contrary, there is plenty of room for further investigation, seeing that so few facts have as yet been ascertained from the small amount of pathological material that has been available in the past.

Diagnosis.—There is little chance of a well-developed case of this kind being mistaken for any other malady, as the affection can usually be recognised at a glance. Several conditions, however, bear a superficial resemblance to facial hemiatrophy. Among these are a congenital inequality of the two sides of the face, the facial asymmetry associated with congenital wry-neck, atrophy of the facial muscles as a result of poliomyelitis affecting the facial nucleus, the asymmetry of the face consequent on facial hemihypertrophy, that present in infantile hemiplegia, paralysis with atrophy in the distribution of the facial and trigeminal nerves, and paralysis of the cervical sympathetic.

In the cases of congenital asymmetry there is not the alteration in the consistence and colour of the skin, nor is there the same difference in regard to the growth of the hair. Where the atrophy is consequent on degenerative changes in the muscles, as in poliomyelitis or other affections of the facial nucleus or nerve, the electrical reactions readily determine with which affection we have to deal in that, as has already been said, the electrical reactions are not altered in facial hemiatrophy, unless it be that the muscles respond more readily to faradism than they do on the unaffected side. When the fifth nerve is involved as well as the facial altered electrical reaction in the muscles supplied by its motor division and the presence of cutaneous anæsthesia make the nature of the case clear. Asymmetry consequent on hemiplegia, infantile or otherwise, is readily distinguished by evidences of hemiplegia in the limbs on the affected side, such as defective power, imperfect development, spasticity, alteration of the reflexes, and the like. Sympathetic paralysis is recognised by the fact that the pupil is small on the affected side, and does not dilate on stimulation of the side of the neck, and there is partial ptosis when the lids are at rest, though the affected one moves as well as its fellow when the patient looks upwards.

Prognosis.—Once developed the affection as a rule progresses, though it does not in the least tend to shorten the duration of life. Spontaneous arrest occurs in some cases, though on what this depends we are ignorant; it cannot be said that therapeutic measures lead to this happy result.

Treatment.—There is no known treatment that has any influence on the course of the affection. Massage, facial gymnastics, and galvanism all deserve a careful trial, and such local measures may be supplemented by the administration of tonics, such as strychnia, arsenic, and iron. In one recorded case a "plumper" of rubber secured to the teeth, but easy to remove for cleaning, considerably improved the deformity of the cheek.

Facial Hemihypertrophy.—This is a rare condition characterised by a general enlargement of the skin, soft parts, and bones of the side of the face, over the area of distribution of the fifth cranial nerve. A few of the cases described seem to have been congenital in origin, but in others the disease developed at or after puberty. Only two cases have been examined post-mortem, and these agree in showing a pure osseous hypertrophy of all the bones on the affected side; also a nodular thickening on the anterior aspects of the frontal, malar and maxillary bones, with a similar thickening of the palate, basilar process, and sphenoid. The soft structures are equally involved, the skin being thickened and coarse, and the hair and sebaceous follicles enlarged.

The cause of this condition is quite unknown; it is doubtful if the theory of exaggeration of "trophic" influence would be substantiated by very detailed observations in future cases.

Facial Nerve, Paralysis of.

See also Brain, Physiology of (*Sixth and Seventh Nerves*); Cornea (*Ulcerative Inflammation, Causes*); Ear, Acute Inflammation of the Middle Ear (*Results*); Ear, Middle, Chronic Suppuration (*Sequelæ*); Electricity (*Galvanism, Treatment by*); Mumps (*Nervous Symptoms*); New-born Infant (*Facial Paralysis*); Parotid Gland, Disorders (*Tumours*); Teeth (*Alveolar Abscess, Sequelæ*).

The facial nerve takes its origin from a nucleus situated in the ventral portion of the tegment at the lower end of the pons. The root fibres at first pass dorsally and loop round the nucleus of the sixth nerve before leaving the pons in close association with the auditory nerve. There is as yet no unanimity of opinion as to whether all the fibres of the nerve are derived from the nucleus whose position has just been indicated; some of the ascertained facts on this question suggest that the fibres which supply the orbicularis palpebrarum and frontalis originate in a nucleus situated in the neighbourhood of that of the third nerve, and that those which innervate the orbicularis oris are derived from the hypoglossal nucleus (*vide* vol. i. p. 470).

Loss of power in the facial muscles may result from a lesion of the centre for facial movements in the cerebral cortex, one which interrupts the path of conduction from this region to the nucleus of the facial nerve in the pons, which involves this nucleus itself, or which implicates the fibres of the facial nerve at some part of their course after they have emerged from their nucleus (*vide* Fig. p. 234). A broad distinction is drawn between the cases in which the facial paralysis is the result of a lesion somewhere on the cerebral side of the nucleus in the pons, and those in which the lesion involves the nucleus or the fibres which spring from it and form the facial nerve. We speak of the former variety of facial paralysis as of cerebral type, and the latter as of peripheral type, and, as will subsequently be seen, there are certain well-marked signs which serve to distinguish the one from the other.

Etiology.—Paralysis of the facial muscles, the result of an affection of the seventh cranial nerve, is in the majority of cases ascribed to exposure to cold, and to this variety the term "rheumatic" facial paralysis has been applied. Sometimes, however, no such exposure can be determined, but in some cases of the kind there is more or less chronic exposure, the patient, for instance, always working in a draughty shop.

Otitis media furnishes a small percentage of cases of peripheral facial paralysis, the nerve being involved in the Fallopian canal (*vide* vol. ii. p. 497). Traumatism supplies a still smaller number of cases, among which are to be numbered those which result during the operation of clearing out the mastoid, those consequent on external blows or wounds implicating the nerve in the neck, notably in operations for the removal of tumours in the parotid region, those the result of fracture of the base of the brain, and such as follow forceps delivery in infants.

Hæmorrhage into the facial nerve or Fallopian canal has been the cause of the paralysis in a few cases. Syphilis is rarely responsible for affection of the nerve outside the cranial cavity. In a few rare cases facial paralysis has been a symptom in tabes dorsalis, and then it no doubt depends on degeneration of the nerve.

Like the other cranial nerves, the facial may suffer in consequence of meningitic conditions at the base of the brain, whether syphilitic or otherwise; it may also be involved in hæmorrhage in this situation, or it may be pressed upon by neoplasms. Apart from such direct pressure the facial, like all the other cranial nerves, appears liable to succumb to some more remote influence when a tumour exists within the cranial cavity, possibly general increase of intracranial pressure, or it may be a neuritis, as yet undetermined, but comparable to that which attacks the optic nerves.

Then again the root fibres of the facial nerve or the nucleus may be implicated by a gross lesion within the pons, such as a tumour, hæmorrhage, or area of necrotic softening, while a sclerotic patch has been similarly operative in disseminate sclerosis. Primary degenerative atrophy of the cells of the facial nucleus is met with as part of a more widespread degeneration in which other cranial nerve nuclei are involved, as for instance the hypoglossal in chronic bulbar paralysis. In the rare cases in which facial paralysis is the outcome of the action of the diphtheritic poison, there may, no doubt, be affection of the whole peripheral neuron, including the cells of the facial nucleus in some cases, but this has not been proved. In very exceptional instances the facial nucleus has been the seat of anterior poliomyelitis.

Finally, facial palsy may be congenital, and a particular form of paralysis of the facial muscles is met with as part of the symptom-complex of the Landouzy-Déjerine type of myopathy.

Morbid Anatomy.—We have very little information as to the true state of the nerve in the ordinary cases of facial paralysis such as are supposed to be due to cold. The view that there is an interstitial neuritis with swelling of the sheath of the nerve and compression of the nerve elements is purely hypothetical, and the only positive facts of which we are in possession are distinctly opposed to such a

view. Minkowski examined a case of "rheumatic" facial palsy and found a true degenerative neuritis with destruction of the myelin sheaths, and without any interstitial change; and Déjerine and Theohari have had a similar experience. So too a case of facial palsy consequent on middle-ear disease examined by Darkschewitch and Tichonow revealed a parenchymatous neuritis without interstitial change; while similar observations have been made by Bikelès and by Flatau.

Symptoms.—When there is complete paralysis of the muscles supplied by one facial nerve there is an inability to perform any facial movements on the affected side. Not only are voluntary movements impossible, but emotions such as crying and laughing equally fail to evoke any movement in the affected side of the face; the difference in the behaviour of the facial muscles on the two sides under such circumstances is very striking and incongruous. In attempts to raise the eyebrows, as in the expression of surprise, or to frown, the wrinkling of the skin of the forehead is limited to the unaffected side, that on the affected side remaining smooth and motionless, except in so far as it may be pulled on from the healthy side. When the patient attempts to close the eyes the lids do not meet, and the eyeball is at the same time rolled upwards, so that the cornea is concealed under the upper lid, while sclerotic is alone visible between the unclosed lids. There is inability to raise the upper lip, as in showing the teeth, and the lip also hangs motionless when the patient smiles, except that the whole mouth is drawn to the unaffected side owing to the unopposed action of the healthy zygomatic muscles.

Although the want of tone in the affected muscles makes the difference on the two sides of the face obvious during repose even in young people, the difference is much more striking in the old, in whom the skin has lost much of its elasticity, and in whom wrinkles and furrows are very pronounced. The loss of elasticity of the skin allows the lower lid to fall away from the globe, and the tears, which are much increased by the irritation of the exposed eye, escape on to the cheek instead of finding their way down the nasal duct, and cause the patient much annoyance. Furrows and wrinkles are smoothed away on the affected side, while those on the unaffected side cease at the middle line. The naso-labial groove is much more shallow or practically obliterated, while the skin of the lower part of the cheek hangs pouch-like, and there is marked drooping of the angle of the mouth on the affected side.

During sleep the eye is only partly closed. Attempts to make a puff, as in blowing out a candle, result in failure, in that there is inability to press the lips together on the affected side, while whistling is equally impossible. On drinking, the liquid tends to escape at the corner of the mouth on the paralysed side, while the mastication of food on this side is much hampered, owing to the paralysed buccinator allowing the food to become lodged between the cheek and jaw. The character of the patient's speech may be a little changed in consequence of the difficulty of articulating labial consonants.

The less obvious defects that are present consist in inability to dilate the nostril on the affected side in sniffing; so, too, persons previously able to move the external ear voluntarily find that they are no longer able to do so, and there is inability to bring the platysma into action, as may be done normally by making the patient depress the lower lip. The paralysis of the stylo-hyoid and posterior belly of the digastric cause no detectable defect.

In consequence of certain fallacious statements that have been made to the contrary, it is necessary to insist on the fact that the palate is not paralysed in an uncomplicated case of paralysis due to a lesion of the facial nerve. Moreover, it must be remembered that mere asymmetry of palate or deflection of the uvula from its mesial position is not proof of unilateral paralysis of the palate, but that only when defect of movement is detected in the absence of cicatricial adhesion consequent on past inflammatory conditions is the diagnosis of paralysis warranted. The existence of paralysis of the palate in conjunction with that of the face indicates that the facial nerve is not the only one affected.

So, too, the tongue, when protruded, may appear to deviate to the side of the facial paralysis, an erroneous impression caused by the asymmetry about the mouth, for there is no paralysis of the tongue in an uncomplicated case of facial paralysis.

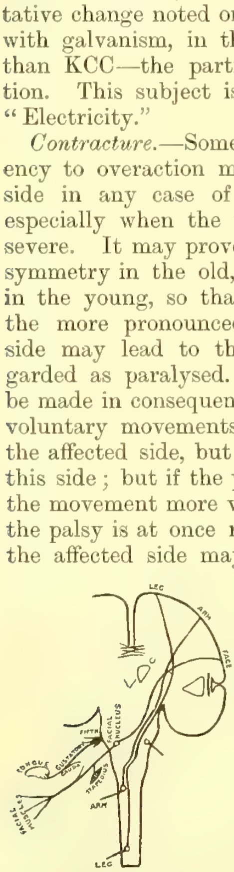
Patients commonly complain of pain on the affected side of the face, and notably in the eye on that side, but there is no blunting of cutaneous sensibility in uncomplicated cases of facial paralysis. None of the special senses are blunted with the exception of taste, which may be defective in certain cases; but smell may appear to be defective on the side of paralysis in consequence of the defective action of the dilator naris in sniffing. Hearing, on the other hand, may become more acute on the affected side, owing to the unopposed action of the tensor tympani consequent on paralysis of the stapedius, so that there is increased sensitiveness especially to musical notes of low tone. Taste is only affected in those cases in which the lesion of the facial nerve is somewhere between the origin of the chorda tympani and the geniculate ganglion, and the loss of taste is then detected on the anterior two-thirds of the tongue on the side of the facial paralysis. Some observers account for the loss of taste by

supposing that there is concomitant affection of the fifth nerve in these cases, and they adduce in support of this the fact that slight blunting of cutaneous sensibility is sometimes present on the paralysed side of the face.

The electrical reactions of the affected muscles are such as are characteristic of a peripheral lesion, so that the reaction of degeneration may be present in typical form (*vide* Electricity). The exact mode of behaviour to electricity naturally varies with the severity of the affection of the nerve, but in a pronounced case the nerve loses its faradic aud galvanic irritability, so that in the course of ten days or a fortnight no response can be obtained on stimulating the nerve with either form of current. In the first two or three days which follow the appearance of the paralysis there may be no loss, or even a slight increase in the irritability of the nerve.

In cases in which the affection of the nerve is less severe there may only be a diminished response to faradism and galvanism when the nerve is excited, and yet there may be a qualitative change noted on excitation of the muscles with galvanism, in that ACC may be greater than KCC—the partial reaction of degeneration. This subject is further considered under "Electricity."

Contracture.—Some contracture and a tendency to overaction may result on the affected side in any case of facial palsy, but more especially when the paralytic defect has been severe. It may prove sufficient to re-establish symmetry in the old, or to produce deformity in the young, so that if viewed during repose the more pronounced furrows on the affected side may lead to the healthy side being regarded as paralysed. This mistake may also be made in consequence of the fact that slight voluntary movements may not only begin on the affected side, but may be more marked on this side; but if the patient is made to perform the movement more vigorously the true side of the palsy is at once revealed, in that, although the affected side may move first, the ultimate result of movement is greater on the unaffected side. The tendency to overaction is well seen in the sphere of one group of facial muscles when the patient brings another group into action. On closing the eyes tightly the zygomatici overact and draw the angle of the mouth outwards, while on smiling, the eye on the affected side is almost closed by the overaction of the orbicularis palpebrarum. Spontaneous twitchings of the affected muscles may occur from time to time, and the reflex excitability of the muscles is increased.



Scheme to illustrate the connection of the facial nerve.

Different Types of Facial Paralysis and their Diagnostic Significance.—In some cases of facial palsy, apparently of peripheral origin, only some of the muscles supplied by the nerve are affected. The muscles which notably escape or suffer much less in degree than others are the orbicularis palpebrarum, corrugator supercilii, and frontalis, and in a smaller proportion of cases the orbicularis oris. It is, moreover, a common experience that in the process of recovery from facial palsy these are the muscles that recover first. Then again, with faradic reaction otherwise abolished in the muscles supplied by the affected facial nerve, there may, nevertheless, be a varying amount of response to faradism in the orbicularis palpebrarum and frontalis, or in the orbicularis oris. These facts detract from the value that certain partial affections of the nerve are supposed to have as indications of the nuclear origin of the disease. There is evidence that though all the facial muscles receive their nerve-supply through the seventh cranial nerve, only some of the nerve-fibres appear to be derived from the nucleus of this nerve, others coming from the nuclei of the third and twelfth cranial nerves. This is suggested by the fact that with affection of the third nucleus the orbicularis palpebrarum has been paralysed in conjunction with the muscles supplied by the third nerve; that with degeneration of the hypoglossal nucleus in bulbar paralysis the orbicularis oris is paralysed in conjunction with muscles supplied by the twelfth nerve; and that in congenital facial paralysis, which is supposed to be due to an affection of the facial nucleus, the orbicularis oris escapes.

Apart from these rare cases just referred to, nuclear facial palsy dependent on degenerative changes in the cells only occurs in association with similar affection of other cranial nerve nuclei, notably the hypoglossal, motor fifth, and oculo-motor.

Pontine Lesions.—As has already been said, lesions of the pons may give rise to facial palsy. When this is the case, other cranial nerves are also affected, as a rule, notably the fifth or sixth, and less commonly the eighth. The facial paralysis that results may or may not be of peripheral type, according to whether or not the lesion is in the lower half of the pons, for it is only when the facial nucleus or the emergent roots of the nerve are involved that this type of paralysis is present. Such cases are usually further characterised by alternate hemiplegia, in which the face is affected on the side of the lesion and the limbs on the opposite side. When the sixth nucleus is also involved there is inability to turn the eyes to the side of the lesion, whether they be also conjugately turned to the opposite side or not.

Meningeal Lesions.—Another place where the facial nerve may become involved in disease is, as has already been said, at the base of the brain, between its exit from the pons and the internal auditory meatus. The most reliable evidence that this is the seat of lesion is the detection of affection of the auditory nerve on the same side, in the absence of middle ear disease. It must not, however, be supposed that the two nerves are always both affected under such circumstances, despite the fact that they be in such close contiguity, for, on the contrary, one or other may be alone affected. Gummatous meningitis is commonly the cause of an affection of the nerve in this situation, but tumours may also be operative, as may fracture of the base of the skull.

Lesions of Cerebral Type.—The paralysis which results from a lesion situated above the facial nucleus, either involving the facial centre in the cerebral cortex, or some part of the motor path between this and the facial nucleus in the pons, is characterised by a defect of movement which is chiefly seen in the lower part of the face, the upper half suffering much less in degree; then again there is commonly a marked difference in regard to the behaviour of the facial movements on voluntary efforts as compared with the effects of emotion, for on smiling or crying the defect of movement is, as a rule, much less evident than on voluntary effort, such as showing the teeth. Moreover, the facial muscles respond normally on electrical excitation, and there are usually signs of similar affection of the limbs on the same side, constituting a hemiplegia of ordinary type.

Facial Diplegia.—Instead of the facial palsy being unilateral as commonly obtains, sometimes both sides of the face are affected. Gummatous meningitis, already referred to as a cause of facial paralysis, may result in facial diplegia, as may fracture of the base of the skull, and in both cases bilateral deafness may be associated with the facial palsy. The diphtheritic poison is, however, responsible for the largest proportion of cases of facial diplegia, under which circumstance many other muscles supplied by cranial and spinal nerves may be involved, notably the palate, ocular muscles, and diaphragm. The ordinary so-called "rheumatic" facial paralysis, already fully described, is occasionally bilateral, in which case one side is usually affected first, and the other subsequently becomes involved. Double otitis media has been known to cause facial diplegia, as has periostitis consequent on teeth extraction and forceps delivery; but in connection with the last class of case it must be remembered that bilateral facial paralysis may be congenital in origin. The facial diplegia met with in the facio-scapulo-humeral type of myopathy is characterised by affection of the orbicularis palpebrarum and oris, in addition to which the zygomatici are usually defective. There is, accordingly, inability to close the eyes properly, the lower lip hangs down and is everted, the patient is unable to pout, whistle, or blow out the cheeks, and on smiling the angles of the mouth are not drawn upwards and outwards as obtains normally when the zygomatici are intact. There is diminished response in the atrophied muscles to both forms of current proportional to the degree of atrophy present, but the reaction of degeneration is not met with.

Prognosis.—The prognosis in facial paralysis is largely influenced by the probable seat of the lesion causing the defect, and its nature. Indications that the condition is due to an affection of the nucleus of the nerve, whether consequent on gross lesions in the pons or on chronic degenerative changes in the cells of the facial nucleus, are of serious import, and recovery cannot be expected. Evidence that the nerve is implicated outside the pons is little less serious, except that when due to gummatous meningitis some improvement may result from appropriate treatment. Prognosis is eminently more favourable when an affection of the peripheral portion of the nerve is responsible for the paralysis, but it naturally varies with the severity of the affection, and to some extent with its cause. When due to otitis media the results are, as a rule, unsatisfactory, even if the ear disease be successfully treated; traumatic cases are usually unfavourable, as are also those in which there is reason to suspect hæmorrhage into the nerve. In the variety due to cold, as indeed in any form due to a peripheral affection of the nerve, an electrical examination affords the best means of arriving at a reliable diagnosis. If faradic excitability be preserved in the nerve at the end of about ten days, recovery may be confidently expected in about a month; if there is notable diminution of faradic excitability at the same period, recovery will probably be delayed until the expiration of about two months; while if there be no response to this form of current at the time already indicated, three or more months usually elapse before recovery can take place. Faradic excitability may not return for two or three months, however, and yet recovery may ultimately ensue; but under such circumstances prognosis ought to be most guarded, and so favourable a result should never be promised. The fact that contracture may be subsequently substituted for paralysis must never be lost sight of, especially in severe cases, a condition which if slight may be recovered from, but which when pronounced is usually permanent, though it may lessen somewhat in time.

Treatment.—Little can be done which is of advantage in the treatment of facial paralysis, except when it is due to a peripheral affection. In any case in which syphilis is suspected, however, mercury and iodide of potassium ought to

be given in full doses, while measures are at the same time adopted for keeping up the nutrition of the muscles until recovery of the nerve has been established. When ear disease is the cause, it is of primary importance to treat that condition, as until it is remedied little good can be expected to result from treatment of the facial paralysis. The cases due to exposure to cold offer the best opportunities for successful treatment. In every case of the kind fresh exposure to cold should be avoided, and when it is not possible for the patient to remain in the house the affected side of the face should be protected on going out of doors. Counter-irritation behind the ear, in the shape of a blister, or mustard leaves several times repeated, is of service. When there is much pain, and the patient is able to remain indoors, hot fomentations to the affected side of the face will give comfort. Little can be expected from drugs administered internally. The view that the condition depended on an interstitial neuritis with swelling of the nerve-sheath and compression of the nerve, made it likely that good would result from the use of mercury and iodide of potassium, but the ascertained facts which have revealed a primary degeneration of the nerve elements without interstitial change do not warrant this belief, nor does it seem reasonable to expect that anti-rheumatic treatment can have any effect on such a state of the nerve.

The most potent agent that we can employ in the treatment of the local condition is electricity. The milder cases do not call for such treatment, but when the affection is more pronounced the use of galvanism, of such strength as to evoke a moderate contraction of the muscles, is a useful measure for preserving their nutrition until recovery of the nerve results. When there is response to faradism this form of current may be substituted for galvanism, but my own practice is to use galvanism so long as I deem it necessary to continue electrical treatment. Though it is difficult to prove the contention, I am inclined to the belief that too prolonged use of electricity, in any form, is apt to favour the occurrence of subsequent contracture, and that the use of faradism is especially liable to lead to this unfavourable result. However this may be, electrical treatment should not be employed when there is any evidence of contracture; but under such circumstances facial massage, combined with warm douching, may prove of service.

Facial Spasm.

The facial muscles may be in a state of spasm, clonic or tonic, as a result of many morbid conditions of the nervous system, this phenomenon being but one of a number of different manifestations which together make up the clinical picture of some disease. The term facial spasm is, however, used in designation of a distinct clinical entity, and when so employed it relates to an affection of the nervous system characterised by spasm limited to the muscles supplied by one or both facial nerves, or in which, while certain other abnormal phenomena may be present, this is the dominant feature in the case. The spasm is, as a rule, clonic, but it is sometimes tonic; it may involve all or only some of the muscles supplied by the facial nerve, and it may occur in muscles which have either previously manifested no abnormality, or been paralysed.

Etiology.—Direct heredity is very exceptional, but a family history of allied neuroses such as epilepsy and insanity in other members of the family is more common. When more than one member of a family has been affected it is possible that diagnosis has been at fault, the real condition present being simple tic or habit spasm. Females are more prone to attack than males, and the idiopathic form of the affection usually occurs in the later periods of life, as between the ages of forty-five and sixty years. A neurotic temperament, previous manifestations of some other neurosis, and a lowered state of nerve tone consequent on some debilitating influence, may all be precursors of facial spasm, and, according to some observers, dominant among the latter conditions is mental emotion. It is interesting to note in this connection that the muscles concerned with emotional movements of the face are those that are most often involved in facial spasm. Another potent factor in causation is some peripheral source of irritation especially occurring somewhere in the distribution of the fifth cranial nerve, notably in the orbital region, which some writers consider a more important cause than emotion. Migraine has been the precursor of facial spasm, as has facial neuralgia, but the latter has been a singularly infrequent antecedent considering that an attack of tic douloureux is commonly attended with contraction of the facial muscles. Exposure of the head and face to cold is sometimes a cause, in which case the spasm may be preceded by facial neuralgia. The source of reflex irritation may be situated at a distance, in which category are to be numbered the cases in which uterine or intestinal irritation has appeared to be operative. Many cases in women commence about the climacterium. The repeated performance of some facial movement appears capable of generating the spasm. Then there are cases in which the spasm commences in the muscles supplied by some other nerve, cranial or spinal,

and subsequently spreads to those under the control of the facial nerve, in the same way that, as I shall have occasion to point out later, spasm beginning in the distribution of the facial nerve may subsequently overflow to muscles presided over by some other nerve.

Apart from the cases in which paralysis has preceded the spasm of the muscles organic disease may be responsible for the affection in several ways. In this category may be included cases in which there is pressure on the facial nerve by a tumour or aneurysm at the base of the brain, so too tumours which cause a degree of pressure on the facial nucleus or the emergent fibres of the nerve, insufficient to cause paralysis, may nevertheless evoke spasm in the muscles supplied by the nerve, and a like result may come about in the case of a tumour or softening in the neighbourhood of the facial centre in the cerebral cortex. Trauma to the head during delivery or subsequent to birth appears to have been operative in some cases.

Symptoms.—The subjects of the affection are attacked from time to time by spasm of the facial muscles which causes much distortion of the features. The intensity and range of the spasm vary in different cases, but a typical attack consists in a succession of clonic contractions which follow each other, at first it may be slowly, and then more and more rapidly, until they culminate in what looks like a tonic stage, which in its turn is followed by a series of clonic jerks which are usually separated from each other by longer intervals than those which constitute the first part of the attack, and the terminal contractions are also commonly of greater excursion. With the increasing interval between the contractions there may be a progressive diminution in the range of movement, but, on the other hand, the last excursion may be as great as any that has occurred at all during the attack. Instead of the clonic element being so definite, in some cases the attack is characterised by a sudden tonus, or if there be clonus it is so rapid as to escape detection, a quivering of the contracted muscles being alone seen, and, moreover, an attack of this kind may terminate as abruptly as it begins without any definite clonus being detected. Instead of the spasm taking either of the forms already described, there is sometimes a single momentary contraction of the muscles which resembles the response to excitation of the facial nerve by a single induction shock, and this phenomenon is repeated at short, either more or less regular, or sometimes irregular intervals.

As has already been said, the whole or only some of the muscles supplied by the facial nerve are affected; when the latter obtains, the orbicularis palpebrarum and zygomatici are those usually in action, and they are nearly always chiefly involved, except that the levator labii superioris may usurp the place of the zygomatici in some cases. The explosion of nerve energy may appear to make its influence felt on all the facial muscles on one side simultaneously, or the first manifestations may be definitely seen in one muscle from which the spasm may spread to others, until the whole of the muscles supplied by one facial nerve are in a state of activity. Not only may the spasm begin in one muscle and subsequently spread to others, but it may be limited to certain of the muscles throughout the attack. The orbicularis palpebrarum is both the muscle in which the spasm most frequently begins, and that which is most commonly affected in the partial forms of facial spasm, the condition being then spoken of as blepharospasm, but other of the facial muscles may be similarly affected alone.

Like the more generalised form of facial spasm, blepharospasm may be either clonic or tonic; the former variety is known as nictitating spasm, and consists in a series of winking movements of the eyelids. When the spasm is tonic in character it may persist for only a few minutes, or there may be no relaxation of the lids for hours, days, or even weeks and months. Some reflex source of irritation is commonly operative in these cases, notably in connection with the ocular branches of the fifth cranial nerve. Trigeminal neuralgia sometimes appears responsible, in which cases pressure on the points which may be tender in trigeminal neuralgia may arrest the spasm. The clonic variety known as nictitating spasm may be only a symptom of simple tic (habit spasm), or it may be hysterical, while in other cases no causal influence can be determined.

The ordinary attacks of facial spasm, in which all the muscles supplied by the facial nerve are affected, are repeated from time to time at varying intervals of from a few minutes to several hours, while the attack lasts a few seconds or some minutes. When fully developed the spasm causes partial or complete closure of the eye, wrinkling of the forehead, with elevation or depression of the eyebrow, exaggeration of the naso-labial groove, drawing of the angle of the mouth outward and it may be also downward, with puckering and drawing up of the skin of the chin on the affected side.

The spasm is usually unilateral, and when bilateral it as a rule begins on one side, and is commonly limited to the side on which it commences for some time before it spreads to the opposite side; moreover, it may only become bilateral in so far as the orbiculares, corrugators, or frontales are concerned,—muscles which may also be involved on both sides without any implication of the muscles of the lower half of the face. The latter muscles are rarely affected equally on the two sides except as part of a general bilateral spasm of the face. Usually, though not invariably, the spasm when bilateral is most pronounced on the side on which it is

of longest standing, and a paroxysm may begin on the side originally affected, or on that more recently involved.

It is rare to meet with evidence of spasm of the stapedius muscle. A rumbling noise in the ear has been regarded as evidence of this, and has been an accompaniment of tonic spasm of the orbicularis palpebrarum. Moos regarded giddiness and conjugate turning of the eyes to one side during a paroxysm of facial spasm as evidence of lowering of pressure in the labyrinth and semicircular canals consequent on spasm of the stapedius.

The tendency to facial spasm is increased by any emotional excitement or facial movements such as in laughing, speaking, or chewing, any of which may serve to evoke an attack. Mental and physical quietude, on the other hand, conduces to freedom from spasm. Cold increases the liability to an attack, while warmth lessens it; so too a bright light acts unfavourably, while darkness has a calming influence on the spasm.

It sometimes happens that the spasm spreads to parts supplied by other cranial nerves and even to muscles supplied by spinal nerves; thus it is that there may be spasm of the muscles of mastication, tongue, and neck, notably of the sterno-cleido-mastoids, and that even muscles of the upper extremity may be involved, as may the muscles of the eyeballs under exceptional circumstances. It is also rare for the muscles of the palate to be affected, but when this has been the case bilateral or unilateral spasm of the palate has been associated with unilateral spasm of the face. The writer is not satisfied that there is sufficient justification for including under facial spasm mixed cases such as those that have been referred to in this paragraph.

Tonic spasm of the face as an independent condition is rare, though cold is supposed to have produced it. This form of spasm is, as a rule, consecutive to paralysis of the facial muscles, or is a part of some more general condition such as tetanus, or it may be an hysterical manifestation. When tonic spasm exists, the face may feel stiff, and there may be considerable interference with voluntary movement of the facial muscles. The palpebral fissure is narrowed, the eyebrow elevated, and the angle of the mouth drawn outward and slightly downward, the latter distortion when bilateral giving rise to the peculiar grin that has been designated the "risus sardonicus."

In the absence of organic disease to account for the facial spasm, loss of motor power in the affected muscles forms no part of the clinical picture, although voluntary movements may, of course, be interfered with by the spasm. So too in the absence of organic disease of the nerve, it and the muscles it supplies usually respond normally to electrical stimuli.

Disorders of sensibility are exceptional, and when present belong to some concomitant condition rather than to the clinical picture of uncomplicated facial spasm. Pain, as has already been said, may be associated with some condition in relation to the fifth nerve, and has then been supposed to be concerned in the causation of the facial spasm. It is only when there has been any destructive lesion of the fifth nerve that any blunting of sensibility has been detected on the face. Apart from the auditory sensation that has been attributed to contraction of the stapedius muscle, no subjective sensations occur in connection with any of the other special senses, though loss of taste on the anterior part of the tongue has been noted where there was reason to suppose that there was damage to the facial nerve.

Pathology. — With the rare exceptions in which a gross organic lesion is responsible for the affection, facial spasm has no known morbid anatomy, so that the malady belongs to that large group of affections of the nervous system which we speak of as functional. Two views are entertained: (i.) some morbid state of the cerebral cortex; (ii.) an affection of the facial nucleus. There are points for and against both views, but my own inclination is rather to regard the facial nucleus as the seat of the trouble in the idiopathic cases. *The cortical theory* obtains support from the fact that organic lesions of the cerebral cortex have been known to generate facial spasm, while those idiopathic cases which have developed after some mental emotion are best explained on this hypothesis. As militating against this theory, however, may be instanced the fact that movements, and not muscles, are represented in the cerebral cortex, so that the strict limitation of the affection in the majority of cases on anatomical lines to the muscles supplied by a single nerve is opposed to the cortical origin of the spasm. Moreover, it is contrary to the best ascertained facts that a centre in the cortex — such as that which presides over the facial movements—should discharge time after time, it may be for years, and yet that the spasm should remain absolutely limited to the anatomical distribution of a single nerve, or only spread so as to involve the muscles supplied by the corresponding nerve of the opposite side. In dealing with this kind of spasm of cortical origin we are prepared rather for a discharge which, though beginning of locally and so limited for a time, nevertheless sooner or later spreads so as to involve in definite order parts other than those presided over by the facial nerve. So too paresis commonly follows local spasm of cortical origin, whereas, as I have already said, loss of motor power forms no part of the clinical picture of idiopathic facial spasm. A further point that has its bearings on the question is, that with the cerebral hemispheres apparently intact gross lesions in connection with the facial nucleus in

the pons, or of the nerve itself, have been responsible for facial spasm.

In support of *the alternative hypothesis* that has been advanced, is the fact that has been last mentioned as militating against the cortical theory, in which connection it may be said that when the facial nerve rather than its nucleus has been interfered with, there is every reason to believe that the spasm, nevertheless, takes origin in the nucleus, which becomes deranged secondarily, for it is contrary to all known facts that irritation of the nerve-fibres should be capable of inducing such definite attacks of clonic spasm as occur in these cases, except through the agency of the nerve-cells of the facial nucleus. Another point in support of this hypothesis is the strict limitation of the spasm, in the majority of cases, to the muscles supplied by the facial nerve. As of some value and in favour of the theory now under consideration, is the fact that reflex irritation in the sphere of the fifth cranial nerve, which is specially related to the facial in reflex action, appears to generate the spasm in not a few cases. It is easier to understand that such peripheral irritations exert their influence on the facial nucleus, rather than that their influence is operative on the facial centre in the cerebral cortex. There are two chief arguments that have been advanced against the hypothesis that supposes the spasm to originate in the facial nucleus: the one supposes such a centre as only capable of discharges which result in tonic and not clonic contraction of the muscles; and the other points to the fact that gross cortical lesions generate facial spasm in some cases, and that the idiopathic cases generated by mental emotion are most readily explained by the cortical theory. In regard to the first of these objections, I venture to submit that there is insufficient proof that a centre like the facial nucleus is incapable of discharges which are manifested by clonic rather than tonic contraction of the muscles; and while I admit the strong force of the second argument, it does not appear to me to be absolutely fatal to the lower centre theory. It may be that gross lesions of the cortex or mental emotion only serve to weaken the inhibiting influence exerted by the cortical centres over the subordinate facial nucleus in the pons, and thus allow of spontaneous discharge of the lower centre, in which secondary changes may further have been induced, in the cases of gross lesions of the brain.

Diagnosis.—The first question to be decided is whether the case is one of true facial spasm or some condition simulating it. Having decided that it is genuine facial spasm, we have next to determine whether the condition is idiopathic or the outcome of organic disease. Among the conditions that may be mistaken for facial spasm is a hysterical affection in which the quivering of the muscles does not, however, in the least resemble what is seen in the genuine condition. A tonic spasm of the orbicularis palpebrarum sometimes occurs in hysteria. The age of the patient, the sex, and the detection of hysterical stigmata of one kind or another, usually serve to distinguish these cases. Chorea, in which there is much facial spasm with little evident affection of the limbs, may lead to error, but the character of the spasm and the detection of some spasm which is always present in the limbs ought to prevent mistakes. As regards the distinctions between the idiopathic variety of facial spasm and that the outcome of organic disease there are several important points to be considered. A history of an antecedent attack of facial paralysis, either alone or as part of a hemiplegia, must of course be inquired for. So too the detection of any weakness of the facial movements, together with permanent contracture and overaction, as well as the paroxysmal attacks of spasm, point to an organic origin of the latter, and the detection of evidences of hemiplegia are of like significance. Much more difficult cases to decide about are those in which the facial spasm is evoked by organic disease in the neighbourhood of the facial centre in the cortex, the nucleus in the pons, or the nerve after its emergence from the pons. One of the most valuable aids to diagnosis in this class of case is the occurrence of weakness of the facial movements which comes on concomitantly with the spasm, or which is of later development and progressive. Search should be made for evidence of implication of any of the other cranial nerves, as the detection of such would lend support to the view that an organic affection was primarily responsible for the occurrence of the facial spasm. When muscles like the zygomatici are alone affected it is probable that the disease is in the cortex, but when the whole of the muscles supplied by the facial nerve are involved it may be impossible to decide whether the lesion is in the cerebral cortex, the pons, or in connection with the trunk of the facial nerve outside the pons.

Prognosis.—Excepting where the facial spasm is a symptom of some organic disease, there is nothing in the nature of the affection itself to cause any anxiety as regards the duration of life; but it proves a constant source of worry and annoyance to the unfortunate victim. The affection is most intractable, as a rule, and usually persists for years or permanently in defiance of all treatment, or intermissions in the spasms may occur spontaneously or as the result of therapeutic measures, but there is usually a return of the spasm after a variable period, and it may ultimately be permanent. The more certain we can be that the spasm has been initiated by some source of reflex irritation which can be removed, and the earlier treatment is commenced, the more favourable does prognosis become, while the more uncertain the

cause and the longer the spasm has been in existence the more unsatisfactory is the outlook.

Treatment. — When symptomatic of some organic disease treatment must, of course, be directed to the condition of which the facial spasm is a symptom. In the ordinary cases of the affection a diligent search must be made for any source of reflex irritation, and, if any such can be found, measures should be promptly taken to remove if possible, or failing this, to palliate it. Decayed teeth must be dealt with, errors of refraction corrected, and so forth. When there is reason to suspect cold as the cause of the facial spasm the affected side of the face ought to be kept warm, in addition to which it is of advantage to bathe the side of the head and face frequently with hot water, and free diaphoresis may be induced with advantage at the outset of the treatment. In addition to the removal or palliation of any cause that can be detected, attention must be directed to the nervous system in the hope of finding some means of rendering the facial neurons less prone to independent discharge. One way of attempting to effect this is by the administration of drugs that are known to have a sedative action on the nervous system, but though, probably, every drug of the kind has been tried, they are as a rule impotent, and no single drug can claim a greater measure of success than the possible cure of one or at most two cases.

Morphia, either alone or in conjunction with atropine, has been more successful than any other drug, but the treatment has to be continued so long that dangers of the morphia habit and its attendant miseries proves a strong deterrent to this mode of treatment. Conium and gelsemium have each been said to do good, but the bromides are practically of no value in the treatment of this affection.

Another way in which the tendency to spontaneous discharge may be lessened is by attempting to improve the nutrition of the neurons in the hope of making them more stable. To this end various tonics have been employed, such as arsenic, zinc, strychnia, silver, iron, and quinine; but the results have been practically no more encouraging than with the sedative line of drug treatment. In many cases the objects of the tonic line of treatment are best attained by isolating the patient, insisting on absolute physical and as much mental rest as possible, liberal feeding, and the employment of general massage and faradism. With improvement of general nutrition it may be hoped that the nutrition of the neurons will also be favourably influenced, and that those of the facial nerve will naturally share in these benefits. Where depressing emotions appear responsible for the generation of the spasm every effort should be made to lessen their influence, and any general impairment of the patient's health must be combated.

Good from electrical treatment directed to the facial nerves and muscles can only be expected to arise when galvanism is employed, and, moreover, only the "stabile" method of applying the current is admissible. A gentle current of not more than 2 milliampères ought to be used, and care should be taken to introduce the current slowly and to shut it off as gradually. Opinions differ as to the best positions for the electrodes, but the practice of the writer is to place the anode over the trunk of the nerve in front of the ear or on its branches individually, and to place the cathode at some indifferent point, as for instance the back of the neck, while towards the end of the application a pole may be placed on each mastoid for a few minutes. Facial massage may be advantageously combined with the electrical treatment in some cases.

Blisters and other forms of counter-irritation behind the ear, while producing no permanent good, seem to be capable of diminishing the spasm for a time, and benefit has been observed after counter-irritation to the cervical spine. When there are tender points in the distribution of the fifth cranial nerve and pressure directed to these spots arrests the spasm, some recommend the application of blisters or the thermo-cautery to these points, while Sir William Gowers advises the hypodermic injection of cocaine at the tender spots. Dr. Weir Mitchell recommends freezing of the cheek for a few minutes by a spray every day or every other day.

Stretching the facial nerve can claim no more success than other modes of treatment that have been tried in this affection, for unless the stretching be sufficiently vigorous the spasm is not influenced, and when the stretching is vigorous paralysis of the facial muscles must of necessity result. The prospects of recovery from the paralysis are good, but with return of power, in cases where the nerve has been temporarily put out of action, the spasm reappears. In some cases it may be less severe than before the operation, but in others it is as severe as ever. The operation of stretching should be supplemented by the administration of sedatives for some months in the hope that the combined treatment may be more effective than either is when employed alone.

Facies.—The appearance of the face (or of the whole body) as indicative of disease or health; thus, the *facies hippocratica* is the physiognomical appearance of impending death ("his nose was as sharp as a pen," Shakespeare, *Henry V.*, Act ii. sc. iii.); the *facies ovariana* is the peculiar appearance of the face in women suffering from ovarian tumours; and the *facies choleritica* is that seen in cholera, etc. *See* Appendix Vermiformis, Appendicitis (*Symptoms*); Gastro-Intestinal Disorders of Infancy

(*Chronic Diarrhœa, Symptoms*); Leprosy (*Facies leonina*); Nose, Post-nasal Adenoids (*Symptoms*).

Factories. *See* Employment, Hours of.

Fæces.

See also Appendix Vermiformis, Appendicitis (*Causes*); Children, Clinical Examination of (*Alimentary System, Examination of the Fæces*); Colon, Diseases of; Diarrhœa; Digestion and Metabolism (*Fæces*); Pancreas, Diseases of (*Symptoms*); Pelvis, Perineum and Pelvic Floor (*Lesions, Tears, Incontinence of Fæces*); Physiology, Food and Digestion (*Fæces*); Pigments of the Body and Excreta; Rectum, Diseases of (*Symptoms, Incontinence of Fæces*); Typhoid Fever (*Symptoms*).

Definition.—By the term fæces we mean all the excreta, whatever their source, which leave the body by the anus. Most healthy persons have a movement of the bowels about once in twenty-four hours. Some, however, without any noticeable departure from health, have two movements per day; while others have their bowels moved only every second or third day.

Characters.—The examination of the fæces is of interest to the physician and to the physiologist. To the former the most important methods of examination are the physical and the bacteriological; the results of chemical investigation have hitherto been chiefly interesting to the latter.

The Physical Examination.—A. *The Normal Fæces.*—The character of healthy fæces varies considerably, chiefly owing to the influence of diet. The principal features are as follows:—

(1) The *amount* averages about 6 or 8 ounces daily.

(2) The *consistence* is such that the motions are "formed," that is to say, they take and retain the shape of the bowel. Fæces retained any length of time lose some of their moisture, and become firmer or even quite hard (scybalous).

(3) The *colour* is mainly due to a pigment called stercobilin, which is believed to be identical with hydrobilirubin. On a vegetable diet the fæces are usually pale. On a flesh diet the presence of hæmatin or substances derived from it (*e.g.* ferrous sulphide) may produce a dark shade of colour.

(4) The characteristic *odour* of fæces is mainly due to skatol bodies.

(5) The *reaction* varies, but is usually alkaline.

(6) The *specific gravity* varies considerably. Usually fæces float in water, but if they have been long retained in the bowel they may sink owing to loss of gas.

The Normal Fæces of Infants.—The first discharges after birth are called *meconium.* This is semi-solid in consistence, greenish-black in colour, and composed chiefly of concentrated bile and intestinal debris. It is discharged three or four times daily for two or three days. The stools then gradually change in character, and assume the appearance of healthy milk fæces by the fourth or fifth day.

A healthy suckling discharges two or three ounces of fæces daily. These are of soft, homogeneous consistence, of a rich yellow colour, and of an acid reaction. They have a faintly sour but not disagreeable odour. The colour is due to bilirubin. The reaction is ascribed to lactic acid or to fatty acids. When an infant is fed on cow's milk the fæces are paler, firmer, and neutral or even alkaline in reaction owing to the decomposition of casein.

Casein is always present, and may be present in large amount in the stools of infants fed on cow's milk. Starch, recognised by the blue reaction with iodine, or by the microscope, is always present if the infant is taking a food containing starch, and is more abundant the younger the infant. Fat is present as fat, fatty acids, or soaps. It must not be confused with particles of casein.

B. *Abnormalities in the Fæces.*—(*a*) Abnormalities in the *amount and consistence* of the fæces depend chiefly on activity of peristalsis. If the intestinal peristalsis is either too sluggish or too active the patient will suffer from constipation in the one instance or from diarrhœa in the other. The diet influences the amount and consistence of the fæces very greatly. On a purely vegetable diet the total bulk of the fæces may be increased to nearly three times, and the solid constituents to nearly twice the amount resulting from an ordinary mixed diet.

(*b*) *Colour.*—The fæces may be pale or clay-coloured in any condition which prevents the entrance of bile into the intestine. Such acholic stools may contain an abundance of fat, chiefly in the form of the fatty acids. Abundance of fat is more characteristically seen in cases where the pancreatic juice is absent. Depth of colour may be due to abundance of the normal pigment, or to the nature of the diet (*supra*). The dark colour of meconium is due to a mixture of bilirubin and biliverdin. When hæmorrhage occurs into the intestine, especially high up, the fæces become black and tarry. A black colour may also be due to certain drugs, especially iron and bismuth. Other drugs which may discolour the fæces are hæmatoxylin (logwood); rhubarb, senna, and santonin (yellow); calomel (green). The green colour, after the administration of calomel, is due to biliverdin.

According to Le Sage the green colour of the stools of infants may in some instances be due to the action of a chromogenic bacillus.

(*c*) Alterations in the *odour* of the fæces are common. Thus some of the diarrhœas of infancy are characterised by the presence of a sour odour; others by the occurrence of the very disagreeable odour due to albuminous decomposition. Such a result may arise in the case of acholic stools owing to the proteids being for a time enclosed in fats, and their digestion and absorption hindered. Later the fats break up into fatty acids and glycerine, and large quantities of indol and other decomposition products are then set free by the breaking-up of the proteids.

(*d*) The *reaction* varies greatly in different pathological conditions, but has not been found to be of clinical importance.

(*e*) *Foreign bodies* may often be noticed in stools. Amongst these may be mentioned—

(1) Bodies swallowed, accidentally or purposely, especially by lunatics or children.

(2) Indigestible articles of food (*e.g.* portions of tendons, skins, fruit stones).

(3) Undigested food (*e.g.* in lienteric diarrhœa).

(4) Mucin, which normally forms the basis of the fæces, may be present in some pathological conditions in great abundance, especially in the condition known as mucous colitis, where it may form skins, or flakes, or casts of the bowel (*see* "Colon, Diseases of").

(5) Pus may be present from ulceration of the bowel, or from the rupture of an abscess into the intestine.

(6) Fat is very abundant in acholic stools, but a considerable amount may be present when the diet is very rich in fat or oil. Concretions largely composed of fatty acids have occasionally been produced as the result of swallowing large quantities of oil.

(7) When *blood* is present in the fæces it cannot always be recognised as such, as the corpuscles are destroyed during its passage through the bowel. Under such circumstances the presence of hæmatin may be demonstrated by a spectroscopic examination, or by the production of Teichmann's blood crystals (by diochloride of hæmatin), obtained by adding to the substance containing blood a drop of glacial acetic acid and a crystal of chloride of sodium.

(8) *Parasites.*—The presence of intestinal parasites is recognised by the passage of individuals or portions of them, or of their ova, in the fæces. The description of these will be found in the article.

(9) *Miscellaneous.*—Under this heading may be mentioned gall-stones, enteroliths, and concretions of various kinds, fragments of mucous membrane, portions of tumours, even gangrenous portions of intestine from an intussusception which has sloughed.

Gall-stones.—The examination of the fæces for gall-stones is important in all cases of supposed gall-stone colic. The fæces should be carefully collected, broken up in water, and strained as in examining for parasites. It must be remembered that even when the symptoms have been very severe, the calculi may be very small. The presence of facets upon the calculus is important as indicating the presence of other stones. (*See* "Gall-Bladder.")

Intestinal Sand.—The occurrence of "sand" in the intestinal discharges is a rare condition. Several varieties are recognised, of which the principal is the passage of large numbers of very minute biliary calculi. In another form the grains are composed of particles of vegetable sclerenchyma such as the little hard bodies so abundant in the pulp of pears.

Quite recently a troublesome form of diarrhœa occurring amongst our troops at Modder River, South Africa, has been attributed to the irritation of the bowel by the quantity of sand swallowed during the prevalence of severe sandstorms.

The Fæces in Certain Diseases.—In a number of diseases the fæces are more or less characteristically altered.

In *typhoid fever* the occurrence of intestinal symptoms is very inconstant. When diarrhœa is present the stools are usually copious, greyish-yellow, and of the general consistence and appearance of pea-soup. The severity of the diarrhœa is no gauge of the extent of the intestinal lesions. Eberth's bacillus cannot usually be found in the stools before the end of the first week.

In *dysentery* the stools vary considerably in different forms of the disease. Commonly they are frequent, gelatinous, and bloody, and if the disease has lasted for some time contain grey or brown shreddy material. In amœbic dysentery the stools are characteristically fluid. They contain much mucus and usually blood, and active amœbæ may be found in them on microscopic examination.

In *simple ulcerative colitis* diarrhœa is a prominent symptom, and the disease is often wrongly regarded as a form of dysentery. The stools are usually frequent, fluid, dark, and foul-smelling. They may contain mucus, shreddy material, and blood which is usually red and fluid, and not intimately mixed with the fæces. Pus, fragments of tissue, and blood are the most valuable signs of intestinal ulceration.

In *cholera* the stools are spoken of as rice-water stools, from their resemblance to water in which rice has been boiled. They are watery, pale, alkaline in reaction, and have a specific gravity of from 1005 to 1010. Before the period of reaction the comma bacillus may be found in pure cultivation in shreds of mucus.

Choleraic diarrhœa occurs also in British cholera and in some forms of irritant poisoning.

In inflammatory affections of the colon *mucus* may be very abundant in the stools, fæcal matter being very scanty. Abundant mucus is specially characteristic of membranous or mucous colitis.

Microscopical and Bacteriological Examination.—There is nothing special in the methods required for the microscopical and bacteriological examination of the fæces. *Meconium* consists of intestinal mucus, epithelial cells, vernix caseosa, bile, hairs, fat-globules and cholesterin. Stains on cloth may be recognised by the peculiar odour, and by the precipitation of mucin on adding acetic acid to a watery extract. In the *milk-fæces* of a suckling may be seen epithelial cells, round cells, fat-globules and crystals, protein substance, mucin, bacteria in great numbers, and occasionally yeast fungi. Fat is normally very abundant in the stools of the infant. The proteids of breast milk are almost entirely absorbed, but when the infant is fed on cow's milk the stools usually contain a considerable quantity of undigested casein. Particles of casein in the stools may be distinguished by the microscope from masses of fat. Starch granules may be recognised if the food contains starch.

On an ordinary *mixed diet* the stools contain distinctly recognisable portions of vegetable tissue, vegetable cells and woody fibres, starch cells, hairs, striped muscular fibre, elastic and areolar tissue, fat chiefly in the form of crystals, cells and mucus derived from the intestinal tract, crystals of different kinds, innumerable micro-organisms, and debris of various origin.

The ova of intestinal parasites may have to be carefully searched for, but may be present in enormous numbers. The continued presence of ova in the fæces after the discharge of a worm indicates the presence in the bowel of other specimens.

Micro-organisms.—At birth the entire alimentary tract is sterile, but within a few hours organisms are present in the discharges. As long as the infant is fed at the breast only two varieties of organisms are constantly present in the intestine. Of these the *bacterium lactis aerogenes* lives in the upper part of the small intestine, and appears only exceptionally in the fæces, while the other, the *bacterium coli commune*, occurs in immense numbers in the colon and in the fæces.

In the adult the fæces possess a very varied flora even in health. It has been supposed by some that the acid gastric juice acts as an antiseptic barrier to the passage of micro-organisms, but it appears that any of the numerous organisms which occur in the mouth or in the sordes of the teeth may get into the intestine. Several observers agree that many of the non-pathogenic bacilli and micrococci are stained of a blue colour by an iodo-potassic iodide solution, a fact of some importance in distinguishing them from pathogenic organisms which they may resemble.

The *bacterium coli commune* is of particular interest because, although not ordinarily pathogenic, it may under certain conditions become so. It closely resembles the *bacillus typhosus* in many of its characters, but may be distinguished by its growth upon potatoes, on which it produces a brown pellicle, while the typhoid bacillus forms a translucent film. A closely allied organism belonging to the coli group is the *bacillus enteritidis* (Gaertner)—possibly not a single species—to which have been attributed several outbreaks of diarrhœa and meat poisoning.

In typhoid fever the *bacillus typhosus* cannot usually be found in the stools before the ninth or tenth day of the fever. (The urine may also be examined for this bacillus.) The *bacillus tuberculosis* may be found in the fæces, but its presence is of no value for the diagnosis of intestinal tubercle, as it is so frequently swallowed.

The *comma bacillus* can frequently be readily found by the direct examination of choleraic stools. The stools should be examined as soon as possible. Some of the choleraic material is spread on several slides, fixed by heat, and stained with carbol-fuchsin. The bacilli are frequently found grouped in little masses, in which the individuals all lie in the same direction, like "a shoal of fish." Cultivations should also be made (in alkaline bouillon).

Amœbæ are very numerous in the fæces in cases of tropical dysentery (amœbic dysentery). The stool should be examined as soon as passed, preferably on the hot stage; or films may be made, fixed with corrosive sublimate, and stained; or portions of the discharge may be fixed and hardened with corrosive sublimate, and cut into sections by the paraffin method.

The Chemical Examination.—The chemical examination of the fæces is undertaken chiefly in connection with experiments on metabolism to ascertain the amount of nitrogen and of fat excreted. Nitrogen is taken as the index of proteid katabolism. It is eliminated chiefly by the kidney as urea and uric acid. The total amount of nitrogen contained in the fæces is estimated and added to that eliminated by the urine.

Fat is usually estimated by counting as fat whatever can be extracted from the fæces by ether.

The constitution of the fæces is highly complex and variable, and tables of quantitative composition are of but little value. Among important substances occurring in the fæces may be mentioned:—

(*a*) *Albuminoids* (collagen, chondrin, keratin, etc.). Normally the stools give no proteid reaction, but albumins may be present in considerable quantity in some forms of diarrhœa,

and in cases where the diet contains an excessive amount of milk or cheese.

(*b*) *Mucin* may be said to form the basis of normal fæces, and in some pathological conditions is very abundant.

(*c*) *Peptones*, although probably absent from normal fæces, may appear in many pathological conditions, *e.g.* in the acholic stools of jaundice.

(*d*) *Carbohydrates* are almost completely absorbed, except when the diet contains considerable quantities of green vegetables or pulses. The stools of young infants who are being fed on a starchy food may contain considerable quantities of starch.

(*e*) *Urea.*

(*f*) *Fat* is almost completely absorbed from an ordinary mixed diet. It may be abundant when in excess in the diet, or when the pancreatic juice or the bile is absent. The stools may contain fat or oil, salts of the fatty acids, volatile fatty acids (acetic, butyric).

(*g*) *Aromatic substances* derived from the decomposition of proteids (phenol, indol, skatol, etc.).

(*h*) *Bile acids.*

(*i*) *Pigments.*—Stercobilin (hydrobilirubin). Blood pigments and derivatives: hæmatin, hæmatoidin (rare), ferrous sulphide. Bile pigment (not in normal fæces except in children).

(*j*) *Cholesterin.*

(*k*) *Excretin*, a crystalline body described by Marcet.

(*l*) *Mineral Salts* derived from the food, especially salts of the alkaline earths.

Methods of Chemical Examination.—The method of carrying out a chemical examination of the fæces can only be indicated very briefly :—

1. The fæces are to be weighed, then mixed thoroughly with water.

2. Distil off one-third of the volume of the fluid obtained. The distillate is then to be examined for phenol, indol, skatol, and free fatty acids.

3. The residue left in the flask is evaporated, acidified with sulphuric acid, extracted with alcohol, and then with ether, and filtered. The extract contains fatty acids, fats, cholesterin, bile acids, sometimes glucosides, and colouring matter.

4. The residue left after extraction contains keratin, elastin, mucin, nuclein, cellulose, starch, and gummy substances. Should hæmatin be present it will be extracted by the acid, alcohol, and ether, and can be examined spectroscopically. (*See* Hoppe-Seyler's *Handbuch.*)

Fæces, Incontinence of.—This term may be applied to any condition in which the contents of the rectum are discharged involuntarily, but a careful distinction must be drawn between those cases where the lumbar centre which controls the sphincter ani is intact, the lesion being at a higher level, and those where it has been destroyed (*see* vol. ii. p. 383). The distinction can be readily made by passing the finger into the rectum. If the lumbar centre is intact the sphincter will be felt contracting on the finger. In these cases involuntary discharge of fæces only takes place when the contents of the rectum accumulate sufficiently to overcome the sphincter.

When the lumbar centre is paralysed the sphincter offers no resistance to evacuation of the rectum, but in this case also the discharge will take place only at intervals whose frequency will depend on the activity of peristalsis.

Incontinence of fæces may also occur in coma, in the typhoid state, and in conditions of extreme exhaustion. It may result from certain operations, temporarily after stretching the sphincter, permanently after removal of the rectum and the sphincter for malignant disease. Permanent incontinence has accidentally resulted from excision of piles. In highly nervous children a form of incontinence, associated with great feebleness of the sphincter, is met with. These cases are very rare.

Treatment.—In most cases the only treatment possible is careful attention to cleanliness. Special care should be taken to guard the patient from attacks of diarrhœa. If the patient is able to go about some sort of retentive apparatus must be worn.

In the form mentioned as occurring in nervous children, ergot given by the mouth and in suppository has been found successful by Fowler (New York). If this should fail Holt recommends the local injection of strychnine.

[*Schmidt's Method of Examining the Fæces.*—Microscopic examination of the fæces of a patient who has purposely been placed on a specially selected diet may yield some information as to the gastro-intestinal function. This method has been elaborated by Schmidt, whose name it bears. The diet chosen consists of cocoa, milk, eggs, rusks, gruel, butter, underdone minced meat, and mashed potato. A dose of charcoal is given at the commencement of the course to show, by its colour, when the diet is beginning to affect the stools. The fæces are then diluted and examined microscopically for connective tissue, muscle fibre, carbohydrate, and fats. On the above diet the only residue normally found is a few partially digestive muscle fibres, a little potato debris, and some fat. Connective tissue implies inadequate gastric digestion (hypochlorhydria), excess of meat fibres disturbance of the function of the upper part of the gastro-intestinal tract, and large masses of carbohydrates point to defective digestion or assimilation in the small intestine.]

Fainting. *See* Syncope.

Faith Cure. *See* Eddyism.

Falcadina. *See* VENEREAL DISEASE (*Syphilis, Allied Diseases*).

Falling Sickness. *See* EPILEPSY.

Fallopian Tubes.

See also ABDOMINAL TUMOURS, DIAGNOSIS (*Fallopian Tubes*); BROAD LIGAMENT, DISEASES OF; COLIC (*Diagnosis, Fallopian Tube Colic*); GENERATION, FEMALE ORGANS OF (*Fallopian Tubes*); GYNECOLOGY, DIAGNOSIS IN; OVARIES, DISEASES OF THE; POST-MORTEM METHODS (*Abdominal Pelvis, Fallopian Tubes*); STERILITY (*Causes*); UTERUS, MALFORMATIONS OF THE (*Rudimentary Fallopian Tubes*).

Anatomy of the Fallopian Tubes.—The tubes are developed from Müller's ducts, and are therefore to be classed with the uterus. The outer extremity of each duct splits and opens into the peritoneal cavity, so as to form the ostium. The inner limits of the tube are at the corresponding uterine corner, the tubal canal passing through the uterine walls to open into the uterine cavity. There is occasionally a diverticulum in this "interstitial" portion of the Fallopian tube. Between the corner and the outer third of the tube is its "isthmus," whilst the outer third itself forms the "ampulla." These three terms are much used in reference to tubal gestation.

The broad ligament is reflected over the upper border of the tube so as to make a true mesentery ("mesosalpinx," *see* "Broad Ligament, Diseases of"). The peritoneum forms the serous coat of the tube, but does not invest it inferiorly, so that the muscular coat comes in contact with the connective tissue in the mesosalpinx. Hence in tubal pregnancy the fœtal sac may rupture into the tissue without tearing through the peritoneum. The middle coat of the tube is made up of plain muscle-cells, circular in its deeper and longitudinal in its outer layers. These layers are well and regularly developed in healthy young women, whilst in chronic inflammation, with or without obstruction, they sometimes undergo partial and very irregular hypertrophy. When a fœtus develops in the tube the muscular coat never undergoes the uniform evolution which occurs in the uterine walls in normal pregnancy; it always thins and yields, or else expels its contents very early. The inner coat of the tube is its mucous membrane; there is a considerable amount of submucous connective tissue with large vessels, and sometimes a little fat. The mucous membrane is raised into numerous longitudinal folds or "plicæ," often bifid. They end by protruding at the "ostium" as the "fimbriæ"; one fimbria, the "ovariam," runs on to the ovary. The epithelium is columnar and ciliated.

Physiology of the Fallopian Tubes.—The cilia of the tubal epithelium wave from the ostium towards the uterus, and it must be remembered that the cilia of the uterine epithelium wave in the same direction, that is, from the fundus towards the cervix (Hofmeier, Mandl), contrary to what was once thought. Authorities are not quite sure as to the precise changes which occur in the tube throughout the menstrual cycle. The development of traces of decidual tissue under the mucosa in pregnancy seems to be an abnormal or pathological condition; Clarence Webster holds that it is the immediate cause of tubal pregnancy, the impregnated ovum settling on the abnormal decidua, and developing as in the uterus.

The tube certainly transmits the ovum. The theory that the ovum is never normally impregnated until it has left the tube and enters the uterus seems based on an assumption. Spermatozoa enter the tube normally; indeed they have been found in the human Fallopian tube. The downward movement of the cilia do not prevent them from passing up the tube. Impregnation, it would appear, may take place at any point between the ovary and the uterus. The oösperm or impregnated ovum may settle on any point of a perfectly healthy tubal mucosa; putting aside the decidua question, noted above, as unsettled, the theory that the oösperm, arrested in the tube, cannot develop except where the epithelium has been destroyed by inflammation (Lawson-Tait) is an error. Pregnancy may occur in an imperfectly developed uterine cornu, bearing of necessity a Fallopian tube with a normal canal, but cut off from the canal of the cervix. It is clear that in that case the impregnating sperm-cell must have passed along the cavity of the opposite cornu, and then entered the opposite tube and impregnated an ovum in the opposite ovary (transmigration of ovum), or some believe that the sperm-cell may reach the ovary on the side of the undeveloped cornu, and manage to impregnate one of its ova. This idea seems very theoretical. For further information on abnormal impregnation the articles on "Malformation of the Uterus" and "Extra-uterine Pregnancy" should be consulted.

Injuries of the Tubes.—If the Fallopian tube be severed without injury to the structures in its immediate neighbourhood the danger from hæmorrhage is trifling, whilst there is always risk of at least local infection from the tubal mucous membrane. A surgical wound inflicted in an uncomplicated ovariotomy usually heals well as the tubal mucosa is healthy. On the other hand, after the removal of a suppurating tube, unhealthy mucous membrane is exposed on the cut surface of the tube. Hence troublesome adhesions to intestine, or abscess, may develop around the stump. To avoid such complications suspicious mucosa should be touched with pure tincture of iodine or a crystal of carbolic acid. In a few cases the present writer has treated a suspicious tube like an inflamed appendix. The serous and muscular coats are cut through, and then the mucosa is divided closer to the uterine cornu. The cut edges of the outer coats are, lastly, united with sutures so as to close the raw surface. Unfortunately the tissues of the tube are often in such cases too friable and soft to allow of this manœuvre.

Malformation of the Fallopian Tube.—One tube is sometimes absent, but in uterus unicornis it is often found almost perfect on the side where one cornu of the uterus is suppressed. Sometimes a perfect tube opens into a pregnant cornu which does not communicate with the cervix, so that sperm-cells or fertilised ova must have passed from the opposite appendages. *Accessory tubes*, with or without a previous canal, are not rarely seen hanging from the upper or lateral aspect of the Fallopian tube; they bear rudimentary fimbriæ. Pedunculated bodies with fimbriæ, but no canal, sometimes sprout from the mesosalpinx. Kossmann not only insists that they are of Müllerian origin, that is to say, tubal, but also that papillary tumours of the broad ligament are derived from them and not from the parovarium. An *accessory ostium*, with large fimbriæ and a short canal opening into the tubal canal, is a conspicuous object often reported and figured. It may explain some of the phenomena of ectopic gestation, but precise *data* are wanting. Tubal pregnancy and cases where transmigration of ova must have occurred seem rarely associated with accessory ostia.

Atrophy.—The normal senile changes in the tube described by Ballantyne and Williams and by Schnaper may set in prematurely. General atrophy is seen in wasting diseases. A segment of the tube may atrophy when involved in the torsion of an ovarian pedicle, or in chronic perimetritis when a tough band has pressed on it for some time. Under both these conditions the tube may be completely severed. The familiar stretching of the tube over a large broad ligament cyst does not necessarily involve atrophy. If after enucleation the operator leaves the capsule, the tube at once shrinks and may resume its functions.

Hypertrophy.—This condition is seen in pregnancy and often in association with uterine myoma. It must be distinguished from œdema frequent when the tube is attached to a malignant or inflamed ovarian tumour.

Patency and Foreign Bodies.—Something will be said on the former subject under "Catheterism of the Tube." Needles and other foreign bodies have entered the tubal canal. Tubal calculus is a result of salpingitis. Cullen's specimen was one inch in length and S-shaped.

Hernia of the Fallopian Tube.—The tube alone has been found in a hernial sac. This condition probably represents the first stage in ovarian hernia, as Wiart detected it in the body of an infant aged six months, and a second very similar case has been reported. Wiart found that the tube was invested with a mesentery simply derived from the sac itself. In other cases carefully described the tube lay in the sac, but otherwise independent of it, the mesosalpinx having been dragged out of the abdominal cavity with the tube. Over a dozen cases of inguinal tubal hernia have been collected. Boudin reports a case of strangulated femoral "salpingocele." The patient, aged 50, had been subject to femoral hernia for five years. It suddenly became painful, without vomiting or other gastro-intestinal symptoms. Jaboulay of Lyons operated. The Fallopian tube was found strangulated by the neck of the sac, which did not contain either the ovary or any other structure. In Boeckel's case, where a radical operation for the cure of inguinal hernia was performed on a reputed male, a bicornute uterus was found in the sac with a true Fallopian tube and a genital gland which proved on microscopical examination to be a testicle. This case recalls another recorded by Dr. Thomas Chambers, where a reputed female underwent operation for double inguinal hernia. Two bodies like ovaries were removed, but they proved to be ill-developed testes. There is no notice of any Fallopian tube. Esmarch and others have had similar experiences. For further details see Ovaries, Diseases of (*Hernia*). The tube does not always follow the ovary into a hernial sac.

Inflammation: Salpingitis.—This disease is closely associated with perimetritis and inflammation of the ovary. When most clearly "primary" it arises from infection of the mucous membrane derived from the lower part of the genital tract. Hence salpingitis is often seen as a late complication of abortion, labour, and gonorrhœa. In the virgin, salpingitis is sometimes distinctly associated with tubercle; indeed tuberculous disease of the tube has been detected in infancy. But it is reasonable to suppose that chronic leucorrhœa in a sickly or uncleanly subject may end in infection of the tube.

Varieties of Salpingitis.—The classification of

the varieties of this disease is of necessity highly artificial. But the forms of salpingitis present confusing variations. Hence some kind of arrangement of the different types of inflammation of the tubes is necessary and convenient.

Bacteriological Classification. — Salpingitis cannot be safely classified according to the microbe that causes or is supposed to cause it. Authorities on bacteriology are by no means agreed as to the part which germs play in diseases of the genital tract. *Actinomycosis of the tube* may, however, be set apart as essentially a salpingitis caused by a specific organism. In Zeeman's case the right tube was converted into a sac as thick as a finger, full of pus and lined with granulation tissue containing actinomyces.

Classification in relation to Pelvic Inflammation.—Salpingitis is, of course, one of the forms of pelvic inflammation. It has little or nothing to do with parametritis (cellulitis), is often closely associated with ovarian inflammation, and is still more nearly related to perimetritis (pelvic peritonitis). Nevertheless salpingitis appears in different forms which demand special classification. The relation of salpingitis to the more general types of pelvic inflammation will be found described under the proper headings.

Classification according to the Patency or Obstruction of the Tubal Canal.—This is the most convenient classification of salpingitis. It is open to one great objection, for hydrosalpinx is sometimes caused by occlusion of the ostium from without, as will be shown, and may probably develop without any essentially inflammatory changes in the tube. Nevertheless it remains the most convenient and scientific method of classification, especially when the varieties are placed in the following order :—

(i.) *Canal of Tube Unobstructed.*—A. Catarrhal salpingitis. B. Purulent salpingitis.

(ii.) *Canal of Tube Obstructed.*—A. Hydrosalpinx. B. Pyosalpinx. C. Hæmatosalpinx. D. Cystic salpingo-oöphoritis, or inflammatory tubo-ovarian cyst.

Catarrhal Salpingitis.—In this very distinct disease changes are observed in the mucosa such as are familiar to the pathologist in catarrhal affections of other mucous membranes. The epithelium is shed, but not destroyed ; the subepithelial tissue becomes infiltrated. The muscular coat is also the seat of inflammatory exudation, but in chronic cases it hypertrophies. The serous coat usually participates in the inflammatory thickening, yet it is sometimes seen thin and not adherent to the muscular tissue of the tube. When thickened, the change may be due to peritonitis from elsewhere. In recent salpingitis mucus is found in the tubal canal, and may be seen issuing from the ostium. This escape of inflammatory mucus does not necessarily cause peritonitis, as catarrhal salpingitis is rarely if ever septic. In consequence of the infiltration into its tissues the tube is elongated, but being confined in the folds of the mesosalpinx which it does not stretch it usually becomes tortuous. This appearance is characteristic of salpingitis without obstruction, and contrasts strongly with the stretching of the tube and the mesosalpinx over parovarian cysts and with the opening up of the mesosalpinx by the tube when the latter is dilated and obstructed. In chronic catarrhal salpingitis the tortuous walls of the tube become very tough through sclerotic changes. Sometimes earthy salts are deposited in the submucous tissue so that the tube feels gritty. In securing an ovarian pedicle where the tube is affected in this manner the ligature is apt to cut through its brittle walls. The accumulation of mucus in the canal sometimes causes internal obstruction of the tube. Earth salts mixing up with the inspissated mucus a *tubal calculus* is formed. As in chronic cases the hypertrophy of the muscular coat is never uniform, the tube seems knotty. The edges of the plicæ of the thickened mucous membrane often unite, enclosing spaces lined with epithelium. Sometimes warty growths develop on the surface of the mucous membrane. As a rule they are minute and sparse ; but in rarer cases they develop into exuberant masses which take on the characters of a papillomatous tumour essentially innocent though liable after a time to undergo malignant degeneration. Catarrhal salpingitis may continue for a long period without assuming the suppurative type of the same disease.

Purulent salpingitis is sometimes a late stage of catarrhal inflammation, but may without doubt be primary, the discharge being purulent from the first. It may also continue indefinitely without any obstruction of the ostium. On separating inflamed appendages from old perimetritic adhesions it is not rare to see muco-pus exude from the perfectly open abdominal ostium of the tube. In short, purulent salpingitis is not to be looked upon as necessarily the first stage of pyosalpinx. In purulent salpingitis the epithelium is freely destroyed and replaced by granulation tissue. This destructive process is, however, seldom complete, as wide tracts of epithelium are often detected in very chronic cases. The plicæ tend, even more than in catarrhal salpingitis, to unite at their edges and enclose cavities bearing the tubal epithelium. Small-celled infiltration of the subepithelial connective tissue, moderate in the catarrhal form, is marked in suppurative inflammation of the tube.

Closure of the Ostium.—Want of space forbids us to dwell on the more minute pathological changes observed in salpingitis, though it will be presently explained how names have been bestowed on sub-varieties of salpingitis where one particular change predominates. But the mechanism by which the abdominal ostium is

closed must be clearly understood. For long it has been believed that hydrosalpinx and pyosalpinx were simply the later stages of salpingitis without obstruction. Recent observations tend to prove that either condition may be primary. The ostium is obstructed in two different ways. The fimbriæ may be sealed up from without (*perimetritic closure*). On the other hand, the ostium may be closed from within by thickening of the mucous and muscular coats around it, they unite over the thickened and shortened fimbriæ (*salpingitic closure*). This is the manner by which the tube becomes closed in salpingitis. The uterine end of the tube is blocked partly by thickening of the mucous membrane, partly through kinking of the tube against its uterine attachment, or through the pressure of inflammatory adhesions from without.

Hydrosalpinx (drospy of the tube, sactosalpinx serosa) signifies dilatation of the distended tube by a non-purulent fluid other than blood. In some instances it undoubtedly arises from salpingitic closure of the ostium, in other words, as the direct result of salpingitis according to the older theory. In cases of perimetritic closure of the ostium, on the other hand, hydrosalpinx may be primary as far as the tube is concerned, and often not a trace of inflammatory change can be detected in the mucous or muscular coat. The tube dilates greatly, and assumes the form of a retort; as it dilates it opens up the mesosalpinx till it touches the ovary. The epithelium is not destroyed, but becomes flat, losing its cilia. Any form of hydrosalpinx may be infected, usually through intestinal adhesions. Primary hydrosalpinx is often associated with uterine myoma from pressure effects and irritation of the peritoneum, and is then apt to suppurate. A large hydrosalpinx is often mistaken for a tubo-ovarian cyst, as will be explained. Bland-Sutton believes that hydrosalpinx represents an old pyosalpinx, on the ground that hydrosalpinx is sometimes found in one tube and pyosalpinx in the other, but Menge points out that pus can inspissate, but never changes into a serous fluid, whilst the structure of the wall of a hydrosalpinx often shows that it could not have been the seat of suppuration. Catarrhal salpingitis in one tube and purulent salpingitis in its fellow are not unknown; should the catarrhal tube undergo perimetritic closure, the consequent hydrosalpinx might be mistaken for the product of a pyosalpinx.

Pyosalpinx (sactosalpinx purulenta) signifies the dilatation of an obstructed tube by pus. It is a fairly definite disease, and is seen as an almost primary condition. Purulent salpingitis setting in, speedy salpingitic closure of the ostium occurs, the tube then becomes dilated by accumulation of pus in its canal. This pus may be virulent at first, losing its germs after a time, and then it may become reinfected through intestine. Pyosalpinx proper frequently follows childbed and abortion, then it is usually associated with widespread perimetritis, and even the mesosalpinx may become infiltrated. It is this variety of pyosalpinx that is specially liable to burst into intestine or bladder. There is also a distinct gonorrhœal pyosalpinx, with less marked general but as much local change. The infection of a primary hydrosalpinx with pus-producing germs is quite probable. In pyosalpinx, as in unobstructed purulent salpingitis, the tubal epithelium is not always destroyed.

Systematic writers describe several forms of salpingitis according to the preponderance of some particular pathological change. In *salpingitis interstitialis or diffusa* there is great hypertrophy of the subepithelial connective tissue, which tends to cause *sclerosis of the tube*, a common ending of chronic salpingitis, especially in tuberculous disease. *Salpingitis pseudofollicularis* signifies that the fusion of the edges of the plicæ has advanced to an extreme degree. Very large spaces are thus shut in, and the tubal epithelium still lines them; on section they appear like cysts and irregularly-shaped follicles. When this condition is associated with "interstitial salpingitis," the term *salpingitis chronica productiva vegetans* is used by some authors. When warty growths develop as part of the inflammatory process this condition is distinguished by Macrez as *endosalpingitis papillomatosa.* The warts may assume the characters of a papilloma. Marked and unusually uniform hypertrophy of the muscular coat in chronic salpingitis has been ranked as the sub-variety *myosalpingitis productiva*, and is always associated with the "interstitial" form. Chiari's remarkable *salpingitis isthmica nodosa* represents an opposite condition, where the hypertrophy of the muscular coat has been more than usually irregular. Circumscribed areas of hypertrophied or hyperplastic muscular tissue form swellings, sometimes as big as a hemp-seed, in the walls of the tube towards its uterine attachment. They contain cyst-like spaces lined with tubal epithelium, probably involutions of the mucosa. The swellings have been taken for an incipient myoma of the tube.

Hæmatosalpinx and Hæmorrhages from the Tube.—The great majority of cases of hæmorrhage either into the tube or out of its ostium are due to ectopic gestation. The pregnant tube may rupture or may eject the ovum with much hæmorrhage from the ostium (tubal abortion). When the gestation is thus interrupted very early in pregnancy the ovum and products of conception may be overlooked, and the hæmorrhage attributed to some other cause.

The so-called "spontaneous" hæmorrhages from the tube are rare. Bleeding due to hyperæmia of the mucosa in menstruation, in hæmophilia, and in several well-known acute infectious disorders, is not unknown. It seems doubtful

whether blood derived from an unobstructed uterus often escapes through the tubes into the peritoneum, but some pathological preparations appear to prove that such an accident is possible (W. S. A. Griffith, author). Pozzi, at an operation, observed "salpingorrhagia."

By "hæmatosalpinx" bleeding into an obstructed tube, independent of ectopic gestation, is understood. The causes are—gynatresiæ, torsion of an obstructed tube, hæmorrhage into a hydrosalpinx or pyosalpinx, and hæmorrhage from a cancer of the tube.

The hæmatosalpinx associated with hæmatometra is a very definite and exceedingly grave condition. It almost always, if not always, implies that the atresia is acquired and not due to congenital malformation (Kleinhans), and the blood in the tubes is full of septic germs, at least when first shed. The consequent danger is well known.

Torsion of the tube in association with a tumour or inflammation is liable to cause hæmorrhage into the tubal walls, but much blood may also escape into the canal, and as that is obstructed a hæmatosalpinx develops. A "blood tube" of this class has been observed when the opposite tube was the seat of ectopic pregnancy.

Hæmorrhage sometimes occurs in the cavity of a hydrosalpinx. This may be termed "hæmatosalpinx" proper, or "sacrosalpinx hæmorrhagica," though Martin, who coined the term, applies it to nearly every form of hæmatosalpinx. The source of the blood has been much disputed. Some authorities think that hyperæmia of the mucosa, or straining, or local injury, sufficiently accounts for it. Others trace it to a physiological tubal menstruation; if so, they still fail to explain why hæmatosalpinx proper is so rare, whilst hydrosalpinx, guiltless of a drop of blood, is so frequent. Others believe the blood is derived from the uterus—the "reflex" theory still accepted by Olshausen, Sänger, and other living authorities. Altogether the first theory seems most in accordance with the experience of operators. Bleeding may certainly occur into a pyosalpinx. Unusually free bleeding of a ruptured Graafian follicle into the ostium of the tube is rare.

In a case of cancer of the tube described by the author, the growth had bled freely into the cavity of tube, which contained large clots.

Hæmatosalpinx remains, on the whole, an obscure subject deserving further precise study. Winternitz has recently written a good treatise on pelvic hæmatocele, where this question of hæmorrhage from the tube, independent of ectopic gestation, is duly considered.

Tubo-ovarian Cyst.—This term is meant to signify a cyst of the ovary communicating with a hydrosalpinx. It has fascinated many workers. In the author's opinion the greater number of cysts so called are not ovarian at all. This class of cyst may conveniently be divided into (1) pseudo-tubo-ovarian cysts, the commonest kind; (2) inflammatory tubo-ovarian cysts; and (3) Bland-Sutton's teratological tubo-ovarian cysts or ovarian hydrocele.

Pseudo-tubo-ovarian Cysts.—In many museums fine preparations of "tubo-ovarian cyst" may be seen. The tube seems to open into a big cavity bent underneath it at a very acute angle. Fimbriæ appear to expand around the orifice of the tube and to float in the cavity of the cyst. The wall of the cyst bears on its inner side, towards the uterus, more or less ovarian tissue connected with the uterus by the ovarian ligament. Careful inspection of a series of such specimens shows, however, that the ovary is not cystic at all. The cyst is a large hydrosalpinx. The outer half has become sharply bent under the uterine half of the tube, and greatly distended, so that its plicæ are effaced, though traces of them may often be detected on careful inspection. The plicæ of the uterine half, which is less distended, project around the orifice communicating with the dilated portion, assuming the appearance of fimbriæ as they float in the fluid contents. These "fimbriæ," however, can sometimes be tracked along the inner wall of the dilated portion, where they are flattened by pressure, especially if the cystic tube be laid freely open, so as to remove the pressure which stretches out the mucosa. The ovary is not really part of the cyst, but has become adherent to it, and then flattened out between it and the uterus.

(2) *Inflammatory Tubo-ovarian Cysts.*—In chronic inflammation of the tube and ovary cystic degeneration of the latter is frequent, the cysts being probably dilated follicles which cannot burst externally. Hydrosalpinx and pyosalpinx being exceedingly common under the circumstances, it is not surprising that the cystic tube may communicate with the cystic ovary. The mesosalpinx is always opened up when a cystic tube is large, so that a tube lies in the closest contact with the ovary. Gottschalk has described a case where the ovary was converted into a unilocular cyst, whilst the fimbriæ of an elongated and dilated tube opened into the cavity of that cyst. It is possible that this case should be placed under the pseudo-tubo-ovarian cysts above described. In bad cases of pyosalpinx the suppurating tube sometimes communicates with a suppurating cystic cavity in the ovary. The infection may be simultaneous, derived from intestine or elsewhere, or the tube may have suppurated first; that is to say, a pyosalpinx may burst into the cyst in the ovary. But these cases are very rare, so that it is difficult to draw safe conclusions about infection, and we must distrust second-hand reports, so easy is it to mistake a dilated tube for a tubo-ovarian cyst.

Hydrosalpinx communicating with a true ovarian cystic tumour should come under the

same class, as it seems to imply inflammatory change even if only affecting the tube. This condition has been reported; not only more than one glandular cystoma (Olshausen, etc.), but also a papillomatous cystoma (Terrillon), have been seen communicating with a hydrosalpinx. Such specimens are rare, firstly, because in ovarian cystic disease the tube and mesosalpinx usually escape inflammation; and, secondly, because when a hydrosalpinx comes to lie on the surface of an ovarian cystic tumour, the walls of the latter are usually thick, and the chances of perforation at the point of contact small. The author has observed several specimens of this condition in his own practice, and in all there was no trace of thinning of either the tubal or the ovarian walls where they adhered. When the tube and the ovarian cyst actually communicate, the latter may discharge externally through the tube and uterus (*Hydrops ovarii profluens*, Hildebrand and others).

(3) *Ovarian Hydrocele.*—Bland-Sutton distinguishes a special variety of tubo-ovarian cyst which is congenital and in no way due to previous inflammation. He goes so far as to make his variety into a separate class, saying that "ovarian hydroceles must not be confounded with tubo-ovarian cysts." In ovarian hydrocele the tube, it is asserted, opens into a recess on the posterior surface of the mesosalpinx known as the "ovarian sac." This recess is a mere pit in the normal mesosalpinx in women, but in "ovarian hydrocele" it is a perfect pouch as in some rodents, and Sutton supports his theory, which is fully demonstrated in his work on *Surgical Diseases of the Ovaries and Fallopian Tubes*, by showing that intermediate degrees of development of the ovarian sac are to be found in other mammals. The ovary projects into the sac. Ovarian hydrocele is certainly a rare condition, many alleged specimens being, in the author's opinion, pseudo-tubo-ovarian cysts; the observer falling into a natural error, explained above, about the fimbriated extremity.

Hydrops tubæ profluens. — Free discharge from the uterus and coincident disappearance of a swelling on one side of the uterus have been observed by authorities of a past generation. The disappearance of a pelvic swelling is a necessary factor as proof of this condition, for in pure uterine catarrh a steady discharge of watery serous fluid is sometimes observed, and at times it may suddenly escape in considerable abundance. The swelling which disappears may, again, be in part a cystic ovary (Hydrops ovarii profluens: see paragraphs on Tubo-ovarian Cysts) communicating with the tube. In Doléris's case of papilloma of the tube, where the ostium was closed and the uterine end open and dilated, as proved at the operation, great quantities of fluid came away; in short, this is one of the best-attested instances of Hydrops tubæ profluens. A pyosalpinx may undoubtedly discharge into the uterus. The author knows of a case where there was a swelling on each side of the uterus, with occasional fever. Operation was declined, and ultimately much pus escaped for weeks from the uterus, the swelling vanishing.

Symptoms and Diagnosis of the Different Forms of Salpingitis and allied Tubal Affections. —Salpingitis comes within the range of the class of disorder known generally as "perimetritis" and "diseases of the uterine appendages," and is closely associated with inflammation of the ovary. The general pelvic symptoms will be found described under pelvic inflammation (see "Pelvis"). An inflamed, unobstructed tube can be felt on bimanual palpation, unless the patient be very fat, but the localising to the tube of any pain that may be present is not possible, nor is it probable that the tube is very tender. The pain probably signifies inflammation or sympathetic irritation of the ovary. In chronic salpingitis, where a history of long-standing pelvic disorder is rarely absent, the tough, tortuous tube is often easy to detect on palpation. Inflamed tubes may certainly discharge into the uterus, but the source of the discharge can rarely be determined in any particular case.

Hydrosalpinx and pyosalpinx are clinically far more definite disorders. When there is evidence of past or present acute or chronic pelvic inflammation, the presence of a fluctuating swelling in the posterior or in one lateral fornix usually signifies the presence of an obstructed and dilated tube. The tube is always tender and fixed, or but little movable. As under both circumstances there is a history of pelvic inflammation, it is not possible to decide whether a hydrosalpinx has developed from salpingitis or from pelvic peritonitis sealing up a healthy tube. The author finds that the latter form, detected at operation, is often exceedingly painful, causing more suffering than is seen in bad cases of bilateral pyosalpinx. Indeed, clinical evidence is not always sufficient to distinguish hydrosalpinx from pyosalpinx. High temperature may exist with hydrosalpinx owing to inflammation in the neighbourhood, and, on the other hand, a chronic pyosalpinx is frequently all but painless, and not accompanied by fever. One feature in all forms of dilated tube is a tendency to empty some of the fluid contents into the uterus, so that the swelling becomes smaller and less tense and painful for a time. But conspicuous hydrops profluens is rare and fluid discharges may be uterine. Hydrosalpinx and pyosalpinx cannot always be distinguished from a small ovarian tumour bound down by adhesions in Douglas's pouch. A careful use of the sound will distinguish the fundus of a retroflexed uterus from a dilated tube in Douglas's pouch. The diagnosis of a dilated tube from tubal gestation is not

always easy. Clinical symptoms are confusing, menorrhagia is frequent in tubal as in ovarian disease, and a sharp, recurrent attack of inflammation is, on the other hand, not rarely associated with cessation of menstruation for many weeks.

Hydrosalpinx and tubo-ovarian cysts often run a very chronic course, they may project into the hypogastrium and iliac fossa, but never steadily increase after the fashion of an ovarian or broad ligament cyst. They are nearly always associated with occasional or frequent attacks of pelvic inflammation, and are very prone to suppurate, especially if adherent to intestine or vermiform appendix.

Most probably the uncertain symptoms associated with pyosalpinx arise from the presence or absence of more or less septic germs in the purulent contents. The most virulent bacilli in acute cases die out if the patient recovers; the gonococcus is also of feeble vitality, and never appears to propagate itself in the tube. Hence a woman may go about for years with a pyosalpinx; but it is always liable to become septic from reinfection. Free discharge of pus from the bowel, where the physical symptoms of dilated tube (and not of parametritis) have been detected, is very strong evidence of pyosalpinx. Fatal rupture of a pyosalpinx into the peritoneum is very rare, as the tube is generally cut off from the general peritoneal cavity by old adhesions. The most experienced gynæcologists, we must remember, all admit that accurate diagnosis of the different forms of pelvic inflammation is seldom easy.

Treatment of the Different Forms of Salpingitis and Allied Tubal Diseases.—Salpingitis without obstruction, diagnosed on reasonable grounds, is to be held as a form of perimetritis, and should be treated as such (see "Pelvic Inflammation"). Rest, vaginal douching, and saline purgatives rarely fail to prove beneficial in a first and acute attack. The chronic forms are always accompanied by the more familiar features of chronic perimetritis, such as constant dull pains in the iliac fossæ and occasional attacks of sharper pain. Rest in bed, douching, etc., make an invalid of the patient; she becomes less able to bear the pain from which she already suffers, and grows all the more exposed to recurrences, being constitutionally weaker. When a venereal taint is suspected mercury and iodides seem of service even when the infection is gonorrhœal. This may account for the good results once claimed for these drugs in the treatment of "congestion of the womb." Taylor of Birmingham has recently spoken in strong support of this medical treatment of suspicious salpingitis, but rightly reminds us that the peritoneal adhesions around the tube are not essentially specific. Indeed, such adhesions cannot be dissolved by physic, and as they are well known to be almost constant and the chief cause of pain there is much scepticism about the value of any drug in the treatment of salpingitis. Deliberate operative interference for purely unobstructive salpingitis is not justifiable. But when in the course of an operation a tube is found bound down by adhesions they should be gently separated if possible. This can be safely done if the tube simply adheres to the ovary, to the back of its own mesosalpinx, or to the peritoneum of Douglas's pouch, but adhesions to any part of the intestine are dangerous to meddle with unless quite soft. Omental adhesions are very common and a definite source of discomfort; they should always be separated. The perfect relief which not rarely follows an "exploratory" operation on the appendages is certainly due in many cases to the accidental or intentional separation of adhesions, especially omental adhesions.

The treatment of hydrosalpinx and pyosalpinx involves grave considerations. There can be no doubt that both hydrosalpinx and pyosalpinx may subside entirely under rest and appropriate medication. They appear to empty themselves slowly, the escape of pus has been frequently observed. If, on the other hand, the patient takes no care of herself, the morbid processes advance, the obstruction increases, and with it the pain from tension on the tubal walls. Catheterism of the tube, even if feasible, is uncertain and dangerous, as will be noted in a further paragraph. But intra-uterine medication often aids the emptying of the tube. The endometrium is nearly always diseased; hence if it be treated with antiseptic applications, such as iodoform, bougies, or swabbing with glycerine or carbolic acid, the morbid changes in its substance disappear, and the adjacent tubal mucosa shares in the benefit, so that the obstruction at the uterine orifice of the tube may be overcome.

Surgical interference may be demanded when there are frequent recurrences of pain and fever, with distinct deterioration of health. The operations recommended by different authorities are: removal of the tube and its ovary, as in ovariotomy, vaginal hysterectomy, and removal of the appendages, opening of the tube through the posterior vaginal fornix, and salpingostomy.

The careful amputation of a hydrosalpinx with its corresponding ovary is often followed by complete cure. The same may be said even when both appendages are removed for pyosalpinx. The surgeon must remember, however, that whilst recovery from an operation where a large ovarian cyst is removed means cure, it is otherwise with amputation of diseased appendages. An after-history of at least two years is needed, as the pedicles too often undergo morbid changes. The advantages of this operation are clear. The parts are well seen, especially if the patient be placed in the

Trendelenburg position. The uterus is left behind, and thus the pelvic floor is not weakened. The dilated tube should be emptied by aspiration before any attempt is made to separate it from its adhesions; the author, in the case of a pyosalpinx, takes the further precaution of injecting tincture of iodine into its cavity. During the separation of adhesions sponges should be packed around the collapsed tube. The stump of the pedicle should be treated as recommended above, in the paragraph on Injuries of the Tubes.

Besides the possibility of hernia of the abdominal wall there are certain grave objections to this operation. Septic peritonitis not rarely follows its performance, and though the pus in a pyosalpinx may be absolutely free from germs, it is, on the other hand, never certain that the fluid in a hydrosalpinx is aseptic. Damage to the rectum may also cause sepsis or fæcal fistula. The uterus left behind is often unhealthy. The most usual evil result, however,—a result which often does not become manifest till six months or more after the operation,—is painful inflammatory change in the stump, sometimes ending in suppuration and discharge of the abscess, and perhaps of the ligature as well through the abdominal wound or into the bowel or bladder. In a bad case of double pyosalpinx retroperitoneal hysterectomy is sometimes advisable.

Vaginal hysterectomy with removal of the diseased tube is practised on a large scale abroad. Some of the perils of the abdominal operation above noted are avoided, but detaching a large tube from adhesions more or less out of sight involves considerable danger. Brilliant statistics have been issued, but the majority of cases which make them up would hardly have been submitted to any operative interference in this country.

Altogether, when the abdominal operation appears inadvisable, and the dilated tube bears well down into the posterior vaginal fornix, free opening and drainage into the vagina is the best line of treatment.

Salpingostomy.—This is a conservative operation chiefly advocated by Polk, Skutsch, and Martin. It consists in opening a hydrosalpinx at or near the site of the ostium, so as to restore the patency of the tubal canal. The incision should be made in the long axis of the tube. The exposed mucous membrane is sewn by fine silk to the serous coat around the incision. Langhans objects to this practice, as it leaves a foreign body in the wound liable to set up local peritonitis, which would certainly close the artificial ostium and entirely defeat the object of the operation. But the mere incision may cause quite enough local peritonitis to seal up the new ostium, besides, it is difficult to make sure that the fluid in the tube and the mucous membrane are free from germs. Oderecht removed a tube on which salpingostomy had been performed a year and a half previously; it had become a pyosalpinx. Out of seventy-six salpingostomies by Martin and Mackenrodt conception was noted in the after-histories of four. The complications associated with hydrosalpinx are very indefinite, and hence the merits of salpingostomy are hard to determine even on statistical evidence. It is most likely to be successful when in an exploratory operation for chronic pelvic pain and swelling the tubes alone appear diseased, and are clearly dilated through obstruction due solely to old perimetritic adhesions, the exciting cause of the peritonitis having passed away.

Catheterism of the Tube.—The tube will sometimes admit a sound passed first into the uterus in the usual manner, but deliberate catheterism is both uncertain and dangerous, hence it is to be rejected as a therapeutic or surgical agent in the treatment of tubal disease. The entrance of the sound into the tube has been proved during a laparotomy. Boursier (*Archives clin. de Bordeaux,* May 1892) succeeded, according to his belief, in catheterising the left tube of a woman aged thirty-one several times; but within four months of the first successful attempt the sound could no longer be passed. It is not always possible to make sure that the sound has not really been pushed through the walls of the uterus into the peritoneal cavity, an accident which is of course to be avoided, though, strange to say, it is not necessarily followed by serious complications.

Tuberculosis of the Fallopian Tube.—Of all parts of the female genital tract the tube is the most often affected with tubercle. Kleinhans recently collected some valuable statistics which show that the evidence gained from necropsies on phthisical women corresponds to that which is gained from abdominal operations.[1] Frerichs found the tubes affected with tubercle in 12 out of 96 post-mortems on tuberculous women, whilst of 814 cases of inflammation of the appendages collected by Martin, Whitridge Williams, and von Rosthorn, the tube was tuberculous in 29. Chaffey and Quarry Silcock found tuberculous tubes in two children between four and five years old; both had general tuberculous disease elsewhere. The fact that tubercle of the tube exists in childhood may explain the appearance of hydrosalpinx and pyosalpinx in young virgins. Cullingworth believes that tubal disease in the virgin is generally, if not always, tuberculous. The disease in question is usually bilateral. Whitridge Williams finds that the majority of cases are secondary to

[1] Since the above paragraph was written, Dr. P. D. Turner of Ryde has shown from careful research at the Brompton Hospital that secondary tubercle of the uterus is relatively much more frequent than authorities seem to believe. It has probably been overlooked by others, as it is not so easy to detect as tubercle of the tubes (*Trans. Obstet. Soc. Lond.* 1899).

tuberculosis of other parts, and due either to infection from the blood or from the neighbouring organs, nor is it possible to exclude blood infection in cases which appear to be primary. Yet primary tuberculous disease has been detected. This fact is not disputed, but there has been much debate as to how the primary infection occurs. Some authorities openly admit that primary tubercle of the tube is practically a venereal disease, that is to say, the tube is infected directly by the semen of a tuberculous man, or, as in the case of syphilis, the tubercle bacillus may be introduced by the finger or the instruments of the gynæcologist. Others hold that the bacillus enters the organism elsewhere, without causing changes at the seat of entrance, and settles in the tube. Possibly, as has been asserted, tubal epithelium damaged by the gonococcus cannot resist the invasion of the bacillus, a condition homologous to tuberculous disease of the testis following orchitis after gonorrhœa.

Tubercle of the tube may justly be called tuberculous salpingitis. The germ attacks the tubal mucous membrane, irritates it, and sets up inflammation. There is an acute miliary and a more chronic form. All the characteristic appearances of tuberculous disease of a mucous membrane are to be seen. In the acute form there is free round-celled infiltration and scattered tubercle, giant cells are scarce, as the tuberculous masses break down quickly, but the tubercle bacillus abounds. Extensive destruction of the mucosa takes place, often with infiltration of the muscular coat. In the chronic form the infiltrated plicæ swell, the surface of the mucosa becomes covered with granulation tissue, but complete destruction of the epithelium does not necessarily occur. Through union of the edges of the diseased plicæ cyst-like spaces lined with epithelium may be detected. The tuberculous growth is often exuberant so as to simulate cancer or sarcoma, giant cells are usually detected in considerable numbers, whilst the specific bacilli disappear. The ostium does not always close, it sometimes discharges the morbid contents of the tube, causing local or general tuberculous peritonitis. When it closes, probably through peritoneal adhesions, the tube dilates and a kind of pyosalpinx develops. Opinions differ about this "tuberculous pyosalpinx," as it represents a chronic condition, and it is not always possible to feel sure that the condition does not signify a hydro- or pyosalpinx secondarily infected with tubercle. A tuberculous tube may certainly infect the bowel to which it adheres, and its contents sometimes burst into the channel of the gut. On the other hand, a tuberculous tube does not always go from bad to worse. It may shrivel up, its cheesy contents becoming infiltrated with earthy salts; its diseased walls undergo a kind of sclerosis through free development of fibrous tissue—salpingitis interstitialis (Whitridge Williams). In a case where the writer opened the peritoneum for diffuse peritonitis involving the tubes, with perfect relief for over a year, the patient died of phthisis three years later. Each tube was found cystic like a thick-walled pyosalpinx; the disease was there evidently quiescent. This condition is what has been detected in many bodies of women who have died of phthisis, often without a clear history of pelvic disease. An unobstructed tuberculous tube is extremely tortuous through thickening and elongation of its coats bound down to a certain extent by the mesosalpinx. Its canal contains cheesy matter and broken-down tissue. A "tuberculous pyosalpinx" contains similar material floating in pus; its walls are extremely thick. It is nearly always adherent to adjacent structures, and associated with very evident naked-eye appearances of tuberculous peritonitis.

Symptoms.—When tuberculous peritonitis can be diagnosed by clinical evidence and physical signs, and a circumscribed body is detected in the lateral fornix, that body is almost certain to be a tuberculous tube. Tuberculous salpingitis is seldom acute, dull pelvic pains set in, and a pelvic swelling is detected. When the disease has existed for some time the tube has usually become a pyosalpinx. It can be detected on palpation as a fixed cystic body in the lateral fornix, not always tender, but often painful because of the peritonitis which it sets up. The patient's health is nearly always impaired. Still, the writer had observed advanced tuberculous salpingitis in young girls well nourished and apparently in blooming health. Menorrhagia is notably frequent; possibly some of the blood comes from the tube, or is due to tuberculous endometritis. Amenorrhœa, on the other hand, is not rare, being due to deterioration of health.

Syphilis of the Tubes.—A syphilitic salpingitis has never been accurately determined. Bouchard and Lépine detected gummata, as large as hazel-nuts, in both tubes in a case of syphilis in a woman aged forty. In investigating appearances seen on a diseased tube removed from a syphilitic patient, the observer must remember that salpingitis from gonorrhœa is very probable in such a subject. Ballantyne and Williams detected miliary gummata in the tubes of an infant aged seven months, and, as well as Donhoff, who has recorded a similar case, they seem to have distinguished what may under reserve be reckoned as specific salpingitis.

Echinococcus.—A case of echinococcus of the Fallopian tubes with a very definite clinical history is reported by Doléris. The patient, aged thirty-six, was the daughter of a butcher, and had been married for twelve years to a man in the same trade. She lived on the premises where his cattle were slaughtered and where two bulldogs were kept. Shortly after marriage

hypogastric pains set in, followed by dysuria. A tumour was detected four years later. The patient had been subject to menorrhagia from girlhood, hence uterine myoma was suspected. The tumour continued increasing very slowly for eight years. It then formed a great, hard, elastic, abdominal tumour, very irregular in form and highly nodular; it had invaded the pelvis. The tumour was removed, it did not adhere to the parietes, but the omentum was almost incorporated with it, and there were firm adhesions to the uterus, bladder, cæcum, vermiform appendix, and rectum. The adherent uterine fundus had to be amputated, the rest of the organ being fixed to the parietes after application of the thermo-cautery to the cut surface. The patient made a good recovery. The tumour weighed over 4 lbs; it consisted of the two Fallopian tubes distended to a degree never seen in hydrosalpinx. Each tube was crammed with hydatid cysts. Much of the peritoneum of Douglas's pouch was resected with the tumour; it was infested with minute hydatid cysts, but both ovaries were healthy. From the history we may assume that pelvic peritonitis developed shortly after marriage, that the tubes became sealed up, a hydrosalpinx developing on each side, and that finally each hydrosalpinx became infected with hydatids. It is instructive to note what was the effect of the very slow, steady pressure of the hydatids which gradually developed within the tubes. One tube measured nearly twenty, the other more than twenty-two inches, they ran a tortuous course, and their dilatations were enormous and very irregular. Such elongation and dilatation is never seen in the commoner forms of hydrosalpinx. The tumour, it must also be noted, escaped suppuration though united to intestine by very old adhesions.

Tumours of the Tube.—*Myoma of the Tube.*—A true "fibroid" of the tube is of great rarity, although the tube is mainly made up of plain muscular fibres like the uterus where myoma is so common. Many alleged cases are doubtful. Thus Simpson's and Schwartz's were both pedunculated and very possibly developed from muscular tissue in the mesosalpinx independently of the tube. Several other cases where the tumour was sessile may have sprung from the same source. In other instances a uniform and extreme hypertrophy of the muscular coat, associated with chronic inflammation, has been questionably classed as "myoma." Two reliable authorities, Bland-Sutton and Jacobs of Brussels, have each removed a myomatous tumour clearly developed in the muscular coat of the tube about its middle third; the tumour was in each case of about the size of an orange.

Chiari's salpingitis isthmica nodosa, already noted, represents an inflammatory change on the muscular coat, not a myoma. Small lumps of muscular tissue containing blind glandular ducts have been detected in the same part of the tube and in the adjacent uterine cornu ("adeno-myoma" of von Recklinghausen). They are pathological curiosities, and their precise origin is uncertain; von Recklinghausen traces them to the Wolffian body, Kossmann to Müller's duct.

Cysts of the Tube.—Kossmann is of opinion that the minute thin-walled cysts so often seen between the folds of the mesosalpinx and sometimes under the free border of the serous coat of the tube itself arise from Müllerian, that is to say, tubal elements. The little accessory tubes, which often spring from the surface of the mesosalpinx, sometimes bear similar cysts. Earlier writers attributed the minute cysts in the mesosalpinx to the Wolffian body. The well-known "hydatid of Morgagni," indisputably a tubal appendage, sometimes forms a cyst of an inch or so in diameter with a very long pedicle. Sänger removed two large masses of cysts containing fibro-myxomatous matter from the fimbriated extremity of a tube; each sprang from a long pedicle which was tied and divided, nothing more being removed. The patient, aged twenty-six, recovered and afterwards became pregnant.

Primary Dermoid Tumour of the Tube.—Dermoid disease of the tube has been observed by Pozzi, who figures a case in the third edition of his *Traité de gynécologie*, but gives no clinical notes. The growth is represented as entirely within the tube, except that the tubal walls are perforated superiorly. Large hairs spread through the wall of the dermoid, projecting into the much-dilated canal of the tube. The tumour was made up of skin with hair, sebaceous and sweat glands and fat, but there was neither bone nor cartilage. The ovary is represented as free from disease and in normal anatomical relation to the tube. As for Ritchie and Treub's cases, it is not clear that the new growth really originated in the tube.

Lipoma of the Tube.—Parona once found a Fallopian tube buried in a fatty mass, nearly 3 oz. in weight, which opened up the mesosalpinx. It is not made evident that the tumour began in the tube. Though adipose tissue, it must be remembered, is found under the mucosa in young subjects, a considerable amount of fat, visible to the naked eye, often grows between the layers of the mesosalpinx near the outer end of the tube. It is from this fact that Parona's tumour very probably arose.

Papilloma and Cancer of the Tube.—Primary papilloma and primary cancer of the Fallopian tube are closely allied. The former disease seems to be of inflammatory origin ("papillomatous endosalpingitis" of Macrez). Nine or ten authentic cases have been collected. When the ostium of the papillomatous tube remains open, the secretion pouring out into the peritoneum may set up dropsy (author's case);

when the uterine end remains open, the secretion may escape in great quantities from the vagina (Doléris's case), a form of "hydrops tubæ profluens." A large papilloma of the tube may prove absolutely innocent; on the other hand, recurrence, when the primary growth was histologically innocent, has been reported (Kaltenbach). Primary cancer of the tube seems to be, at least as a rule, a malignant degeneration of a papilloma. About thirty genuine cases have been collected. A hydrosalpinx invaded by a cancer developed in the ovary must not be mistaken for a primary tubal cancer. Papilloma usually develops in the prime of sexual life, cancer towards the menopause. Beyond the development of a pelvic tumour the symptoms of papilloma are very indefinite. In cancer a watery or sanious discharge has frequently been observed. The only treatment for papilloma or cancer of the tube is removal. When a suspicious tubal tumour is bilateral it is safest to remove the uterus as well as the tubes.

Sarcoma of the tube is extremely rare; two reported cases bear the authority of Sänger. A case of *Deciduoma malignum* has been published.

Falmouth. *See* Therapeutics, Health Resorts (*English*).

False Corpus Luteum.—The corpus luteum of menstruation, or the yellow body which forms in the ovary when ovulation is not followed by pregnancy. *See* Menstruation; Generation, Female Organs of (*Ovaries*).

False Membrane. *See* Diphtheria.

Falsetto. *See* Physiology, Respiration (*Voice, Singing*).

Falx Cerebri. *See* Brain, Physiology of (*Anatomical*); Meninges of the Cerebrum (*Anatomy*).

Fames.—Hunger, especially in such expressions as *fames canina* or *bovina*, which signify unnatural or voracious appetite or bulimia. *See* Appetite (*Increase*); Bulimia.

Family Spastic Paraplegia. *See* Paralysis (*Spastic Paraplegia, Familial*).

Famine Fever. *See* Relapsing Fever (*Synonyms*).

Fango.—An application applied locally at a temperature of 98° to 120° F. in rheumatism, gout, neuralgia, muscular paralysis, etc.; it is said to be a mud, containing iron, sulphur, magnesium, and lime, and is obtained from some of the Italian lakes; *fangotherapy* is the treatment of disease by means of it.

Faradism. *See* Electricity (*Magneto-Electricity*).

Farcy. *See* Glanders.

Fareol.—A proprietary preparation, which acts as an antipyretic and analgesic.

Farre's Tubercles.—The superficial nodules of secondary cancer of the liver. *See* Liver, Diseases of (*Secondary Malignant Disease*).

Fascia.

See also Deformities (*Hands and Fingers, Congenital Contraction*); etc.

Definition.—A fascia is a fibrous envelope binding down muscles or enveloping them in different regions.

Superficial and Deep Fasciæ.—Immediately beneath the skin is a more or less closely-woven layer of areolar tissue containing a varying amount of fat, and known as the superficial fascia. It is in parts capable of division by the dissector's knife into two layers. The external of these layers is practically continuous over the surface of the body. The internal layer sometimes dips down and becomes attached to deeper structures, *e.g.* the deep layer of the superficial perineal fascia is attached to the inferior border of the triangular ligament. The deep fascia immediately covers the muscles, and sends processes which invest them and enclose them in partitions. The tendons of muscles assume the expanded form of "aponeuroses," as in the case of the abdominal muscles, where the aponeurosis of the external oblique constitutes the deep fascia of the abdomen.

Structure.—Both superficial and deep fasciæ are composed of areolar tissue. In the former the bundles of fibrous tissue are closely and irregularly interwoven, and it contains numerous fat lobules. In the deep fascia the fine bundles of connective tissue are disposed for the most part in one or two directions, and form sheets of fibrous tissue.

Functions.—The fibrous tissue of the deep fascia is exceedingly strong and tough, and yet perfectly flexible, but it is almost entirely inextensile; and it therefore maintains the parts which it connects in apposition against any severing force short of that sufficient to rupture it, while its flexibility permits of easy motion. Surgically the deep fascia limits or bounds inflammatory changes and pus until the process becomes so acute that the fascia softens and sloughs, and then sudden extension takes place.

Fasciæ in various Regions.—In the head and neck numerous fasciæ are described,—the

temporal, buccal, masseteric, and the cranial aponeurosis. Of greater importance than these and coterminous with them is the deep cervical fascia of the neck, which acquires much surgical interest from its bearing upon deep cellulitis of neck (angina Ludovici), its relations to the deep veins, and the possibility of entrance of air into the veins by dragging upon the fascia (canalisation of veins). It also directs the course and determines the position of spontaneous opening of glandular and of cervical spinal abscess, and being continued in front down to the mediastinum permits the spread of inflammatory exudations in cut throat and tracheotomy. The deep fascia is also contracted in congenital torticollis and in spasmodic wry-neck. In the thorax the pectoral fascia contains numerous lymphatics which are the channels of general infection in scirrhus of the breast. This fascia is also prolonged upwards as the costo-coracoid membrane, which has an important relation to the first part of the axillary artery. In the upper extremity many parts of the deep fascia have received special names, for example, the scapular, the axillary, the fascia of the upper arm or brachial, the deep fascia of the forearm with its annular ligaments, and the fascia of the palm which is the site of Dupuytren's contraction. The muscles of the posterior abdominal regions are bound down, and some take their origin from the fascia lumborum, which together with the psoas fascia determines the course of spinal abscesses. Anteriorly the transversalis fascia has important relations to inguinal hernia, a special prolongation of it on to the spermatic cord being named the infundibuliform fascia. It is continued downwards on the lateral aspect of the abdomen, and is known as the fascia iliaca where it covers the iliacus muscle; while anteriorly a small portion passes beneath Poupart's ligament, forming the deep crural arch and the front part of the crural sheath. On the spermatic cord are found the cremasteric fascia and the inter-columnar fascia which is derived from the aponeurosis of the external oblique. The pelvic fascia, with its derivatives the obturator and recto-vesical fasciæ, are of more anatomical than surgical interest. That firm, strong envelope of the thigh, the fascia lata, acquires importance from the relation of its saphenous opening to femoral hernia, while on the outer side of the thigh its fibres are so strongly developed as to form a tendon-like structure, the ilio-tibial band. Below the knee an investing sheath of fascia is found, forming at the ankles the annular ligaments. The fascia of the leg is prolonged into the sole of the foot as the plantar fascia.

Injuries.—*Rupture, and Hernia of Muscle.*—Both superficial and deep fasciæ are involved in wounds of some depth. The deep fascia is often extensively ruptured and lacerated in contusions and wounds produced by forces of a tearing nature and in compound fractures of the tibia; the fractured ends of bone are sometimes seen protruding through the deep fascia. If the wound be rendered aseptic, healing of the rent in the fasciæ, as of other soft parts, follows, but if suppuration occur, then pus tracks extensively beneath the fascia and travels to a considerable distance between the muscular compartments. Occasionally, even in an aseptic wound, or without any loss of skin and superficial tissue, the rent in the fascia fails to heal, and a hernia of muscle follows. Lejars describes it as occurring after violent contusions, ulcerations of the fascia, and surgical wounds; and Delorme quotes two cases, one of Verneuil's, where a hernia of the tibialis anticus, as large as the last phalanx of the thumb, prominent during contraction of the muscle, and receding when the muscle was relaxed, followed on ligature of the anterior tibial artery. The other case quoted by Delorme occurred in the forearm after the introduction of a seton, and when the hernia was reduced an opening could be felt in the deep fascia. Hernia of the muscles, especially of the adductors, is met with also in cavalry and hunting men. Such a hernia is recognised by its position, its reducibility when the muscle is in repose, its sudden appearance and hardening when the muscle contracts, and in most cases after its reduction a clear, well-defined opening can be felt in the deep fascia. As to treatment, the pressure of an elastic rubber bandage may serve to keep the hernia back, but if this fail, or if the hernia be rapidly increasing and giving rise to trouble, it may be cut down upon, the fascial edges freed, and united over the muscle by a continuous suture.

Diseases.—Inflammation and sloughing occur in the superficial fascia, and frequently extend to the deep fascia, as in the course of cellulitis, when large portions may be drawn out in the form of wash-leather sloughs. Contraction of the deep fascia is best seen in the palm (Dupuytren's contraction) and in the sole of the foot in association with talipes equinovarus and acquired talipes calcaneus. The deep fascia is often the site of gummata, and occasionally of new growths such as fibromata and sarcomata. The writer has met with several cases of true fibroma of the plantar fascia.

Dupuytren's Contraction (Contraction of the Palmar Fascia).—It is an important fact to grasp that in this affection not only is the palmar fascia itself involved, but also its digital prolongations. The male sex is more commonly affected, and the proportion is about twenty males to one female. The fingers on the ulnar side of the hand are more usually involved, the ring finger the most frequently, then the little finger, and often both fingers. But it must not

be forgotten that the contraction may affect the middle and forefingers, and even the thumb. In many cases both hands are affected, but not simultaneously nor to an equal degree. The right hand, however, is not so much more often affected than the left, as the theory of traumatism would seem to imply. Heredity is a distinct factor in its occurrence, about one in four cases presenting such a history. The age of onset is middle or late life. A fair proportion of cases occur in those who use the palm of the hand much in their daily work, *e.g.* carpenters, drivers, gardeners, etc., but Mr. W. Adams has shown that most of his cases were drawn from the professional classes, and in these instances there was one feature common to all, viz. a gouty inheritance. With reference to the causation, the first point is that Dupuytren's contraction is an affection of that time of life when fibroid changes are supervening, and the second point is that most of the cases have a gouty history. The actual onset is marked in some cases by a single injury to the palm of the hand, instances of which are the following quoted by Abbe:—A patient while climbing a ladder pierced his palm with a piece of frozen mortar and dated the onset of the affection from that time; and another patient, a civil engineer, had a long series of stakes to put into the ground and pressed them hard with his palm. The next day he had a sore palm and traced the affection directly back to this date. But more often we find a history of repeated and slight traumata. Ricard and Ricket record cases in syphilitic subjects in which the affection yielded to iodide of potassium. Abbe believes in a nervous origin of the affection, and Mr. William Anderson advocates the theory of bacterial origin in the following words:—"The situation of the initial lesion and the peculiar tendency of the new growth to feed like a parasite upon the tissues in which it spreads and which it replaces, have led me to believe strongly that the active agent of destruction is a specific micro-organism, which gains access to the subcutaneous tissues through accidental lesions of the epidermis, mostly effected by the finger-nails." But until Mr. Anderson's theory is substantiated we must fall back upon the gouty history and slight and repeated traumata for an explanation.

The *morbid anatomy* of the affection is quite clear. It is primarily an induration and then a contraction of the palmar fascia, and afterwards of the skin, the tendons having nothing to do with it. The nature of the change is a fibroid hypertrophy, either in the local form of small fibromata or as a general hyperplasia. Beginning in the deepest part of the palm, *i.e.* above the base of the ring finger, where a puckering of the skin appears, the contraction spreads downwards along the fascial prolongations to the fingers until they are drawn down into the palm. Bearing on the part played by gout in its production, a dissection by Mr. Lockwood, in which he found the fascial contractions incrusted with urate of soda, is of great value. The *symptoms* commence with a feeling of tightness in the palm and ring and little fingers, and the patient finds some difficulty in fully extending those fingers, and later there are some nodular indurations in the palm and considerable neuralgic pain in the hand. The skin becomes adherent, dry, and thickened, and a puckered dimple appears in the transverse crease of the palm. The fingers then become retracted into the palm until the nails may wound it. The affection very rarely becomes spontaneously arrested, but is progressive, and the tips of the fingers touch the palm in about two years from the first onset. The *diagnosis* should be made from congenital contraction of the finger, from flexion of the fingers due to adhesion of the tendons to their sheaths, and from contraction of the fingers, the result of osteoarthritis. But the typical appearances in Dupuytren's contraction are the nodular thickening of the fascia, the adhesion of the skin, and the freedom of movement of the tendons in their sheaths.

Treatment.—When the affection is at all marked the only measure available is operation. Mechanical extension is useless, painful, and harmful. The operative treatment is either by multiple subcutaneous puncture or excision of the affected fascia by the open method. The best subcutaneous method is that practised by Mr. Adams, and consists in the introduction of a fine tenotome between the skin and fascia, freeing the skin first, and then dividing the fascia in several places so as to isolate the portions of it. Thirty or forty punctures are frequently required. The fingers are then brought as far into the extended position as possible without using any force, and maintained there by a well-padded metal splint for three or four days until the punctures are healed. An extension instrument is then applied for fourteen days or longer.

The open method is to be preferred in the case of those who are unable to devote much time to the treatment, such as manual labourers, but it necessitates complete confidence in aseptic measures. The writer has practised it successfully on several occasions and has every reason to be satisfied with it. An Esmarch's bandage is applied, and a longitudinal incision is made in the palm from just below the line of the superficial palmar arch to the root of the finger most affected. If more space is required, a transverse incision is made at either end of the longitudinal, the flaps are dissected back, and all the contracted fascia in the palm is dissected out, taking great care not to wound the nerves. The contracted bands on the lateral aspects of the fingers are divided subcutaneously,

and the fingers strengthened as much as possible without force. Hæmorrhage, often free on account of the vascularity of the part, is then arrested and the wound in the palm closed by horse-hair sutures. In fourteen days the palm is completely healed and the fingers are mobile. The writer prefers the open method.

Contraction of the Plantar Fascia.—Any cause which tends to unduly approximate the heel and heads of the metatarsal bones results in this contraction and gives rise to the so-called pes cavus. The conditions in which this form of fascial contraction are met with are talipes equinus, talipes equino-varus, talipes calcaneus, and some cases of Friedreich's disease. In all but the last it is permissible and often needful to divide the fascia subcutaneously in one or more places, and after fourteen days to stretch the sole.

Fasciola Hepatica. *See* Mouth, Injuries and Diseases of the Jaw (*Parasitic Disease*); Parasites (*Helminths, Trematodes, Fasciolidæ*).

Fasciotomy. *See* Deformities (*Paralytic, Treatment*); Fascia.

Fastigium.—The stage of acme in fevers. *See* Temperature (*Fever, Stage of Acme*).

Fasting. *See* Physiology, Food and Digestion (*Metabolism during Fasting*).

Fat. *See* Arteries, Diseases of (*Fatty Degeneration of Intima*); Blood (*Examination, Methods, Micro-chemical for Fat*); Bone, Diseases of (*Acute Osteomyelitis, Local Symptoms, Fat in Pus*); Contusions (*Clinical Features, Fat Embolism*); Diabetes Mellitus (*Treatments, Fats in Diabetic Diet*); Diarrhœa (*Character of Stools, Fat*); Diet (*Fats*); Digestion and Metabolism (*Absorption of Fats*); Enzymes (*Fat-splitting Ferments*); Fat Embolism; Fæces (*Chemical Examination*); Fractures (*Complications, Fat Embolism*); Heart, Myocardium and Endocardium (*Morbid Processes, Fatty Infiltration*); Hernia (*Femoral, Fatty*); Infant Feeding (*Milk, Quality, Fat*); Liver, Diseases of (*Lardaceous Liver*); Liver, Physiology of (*Functions, Regulation of Supply of Fat*); Lungs, Vascular Disorders (*Pulmonary Embolism, Causes*); Mammary Gland, Diseases of (*Neoplasms, Fatty*); Micro-Organisms (*Bacteria, Fat-splitting Ferments*); Milk (*Examination, Estimation of Fat*); Muscles, Diseases of the (*Muscular Degenerations, Fatty*); Pancreas, Diseases of; Peritoneum, Tumours of (*Fatty*); Physiology, Tissues (*Fat*); Physiology, Blood (*Fats*); Physiology, Food and Digestion (*Energy-Value of Fat, etc.*); Pregnancy, Physiology (*Nutrition*); Sclerema Neonatorum (*Fat Sclerema*); Skin, Anatomy and Physiology (*Origin of the Skin-Fat, Excretion of Sebum*); Stomach and Duodenum (*Digestion, Fats*).

Fat Embolism means the impaction of minutely subdivided oil globules in the smallest arteries and capillaries of the lungs and other organs, with consequent embolic obstruction and its effects.

Pathology and Morbid Anatomy.—In the tissues each fat cell represents liquid matter enclosed in a membrane. These membranes may be ruptured by any injury producing laceration of adipose tissue, the oil escaping in the form of minute globules. These enter the circulation by means of the torn veins, and also by the lymphatic vessels, whether they be ruptured or not. After fractures the absorption of oil globules from the bone marrow is favoured by the increased tension and by the fact that in the bone the torn ends of the veins remain open. Fatal fat embolism, though rare, is almost always due to extensive fracturing of long bones. But the condition probably occurs to a greater or lesser degree after all fractures, and may be seen after amputations, joint resections, crushes, and other injuries of bones. In rare cases it may occur after forcible straightening of deformed joints, abscesses, especially acute osteomyelitis, widespread subcutaneous injuries, and injuries to fattily degenerated tissues, *e.g.* the liver, as in Zenker's case. The condition may also be found in some cases of diabetic coma (Hamilton Starr), but in these cases the fat embolism may be stimulated by post-mortem changes in the lipæmic blood.

In the blood, and especially in that of the pulmonary artery, small clear shining drops of fat may be visible to the naked eye, and the blood has often a peculiar oily appearance. [It is stated that in diabetes the oil globules are combined with granular matter, and more minutely subdivided than when they are derived from bone marrow.] The oil globules, whatever their source, are carried to the right ventricle, and then become impacted as emboli in the finest arteries and capillaries of the pulmonary system. Venous congestion of the lungs occurs in consequence, and there may be hæmorrhages on the surface and in the substance of the lungs, which are usually very œdematous and may be atelectic in parts. In some cases the bronchi are filled with frothy fluid, and the pleuræ contain bloody serous fluid. The other organs are in some cases quite healthy, but owing to fat embolism in them may show venous congestion and capillary hæmorrhages. The brain and kidneys are more often affected than the liver. Fatty degeneration of the myocardium has been observed and ascribed to plugging of vessels and scanty capillary anastomosis, while congestion in the right ventricle is due to obstructed pulmonary circulation.

Histology.—The pulmonary capillaries of most arteries are plugged with oil, which stains black with osmic acid. The oil is seen either in homogeneous masses or else resembles a chain of pearls. In the kidneys the globules are mainly seen in the glomerular coils [less abundantly in the vessels of the convoluted tubules].

Clinical Characters.—From two to four days after the majority of fractures fat may be found as small oily droplets in the upper parts of the specimens of urine. The condition lasts for about two weeks, and is as a rule a harmless complication. In rare cases from one to three days after the injury serious symptoms appear. In addition to the presence of fat in the urine there is dyspnœa, which gradually increases, the face is pale, the pulse feeble, and the temperature low. In some cases there may be hæmoptysis, with dulness over the lungs and audible râles, but in others no pulmonary changes can be detected. Restlessness, cyanosis, and localised muscular twitchings have been described. In the course of a few hours the pulse becomes weaker and more rapid, there is somnolency which increases till the patient is comatose, and usually from five to twenty hours after the onset of the symptoms death supervenes either in coma or from pulmonary œdema.

Diagnosis.—The symptoms, except in rare cases, do not appear until 24-72 hours after the injury, while the onset of shock is rapid. Ordinary pulmonary embolism is usually a later occurrence than fat embolism. Acute uræmia is excluded by the presence of fat in the urine, and by the absence of such signs as albumin and casts in the urine, convulsions, vomiting, etc.

Prognosis.—Although fat embolism may aid in causing a fatal result it is very rarely the sole cause of death. Thus, Mech in 1892 could find records of only seventeen cases where no other cause of death was ascertainable. The gravity of any case depends on the extent of the fat embolism and on the natural powers of resistance of the individual. If those serious symptoms already described should ensue, the prognosis is very grave.

Treatment is of little avail, and can only be directed to the relief of symptoms. Warmth, counter-irritation to the chest, and stimulants—alcohol, digitalis, ether, and oxygen—may be serviceable, especially if the dyspnœa be urgent.

Fat Necrosis.—The occurrence of white patches, size of a hazel nut or larger, in the subperitoneal tissue and in the omentum and mesentery; it is caused by the splitting up of fat (perhaps under the influence of a fat-splitting ferment of the pancreas) into glycerine and fatty acids, the former being absorbed and the latter uniting with lime to form the opaque patches; it would seem to be due in some way to an interference with the secretion of the pancreas and to its consequent passage into the lymphatics or blood-vessels. *See* Pancreas, Diseases (*Pathological Considerations, Fat Necrosis*).

Fatigue. *See* Physiology, Tissues (*Muscle, Factors modifying Contraction*); Physiology, Nervous System (*Brain, Fatigue of Cerebral Mechanism*).

Fats.—Fats are ethereal salts or esters, and from them soaps can be formed (by the action of metallic hydroxides); stearine (glyceryl tristearate) is the chief constituent of hard fats, and it enters largely into the composition of suet (*Sevum Præparatum* of the Pharmacopœia) of cacao butter (*Oleum Theobromatis*); lard (*Adeps*) and benzoated lard (*Adeps Benzoatus*) are also official preparations, and contain a large proportion of olein along with stearine and palmitine; lard, with some of the oils expressed, is used (as *Adeps induratus*) in India and the Colonies. Fats split up into fatty acids and glycerine, but some, such as those obtained from sheep's wool (see *Lanoline* or *Adeps Lanæ Hydrosus*), yield cholesterine instead of glycerine. *See* Adeps; Fat; Lanoline; Prescribing; etc.

Fatty Degeneration. *See also* Fat.—A peculiar degenerate change in the tissues consisting in the formation of fatty granules in the cell cytoplasm; it often follows upon cloudy swelling (*q.v.*), and, like it, is due to the action (longer and more marked) of a toxine, such as that of one of the exanthemata or of septicæmia; it is also found associated with phosphorus or chloroform poisoning, in diabetes mellitus, in anæmia, in paralysed muscle, in atheromatous arteries, and in some neoplasms; the organ affected has a lessened consistence, a yellowish or yellowish white colour, and shows greasiness on section; and the presence of fatty degeneration in a tissue can usually be demonstrated by osmic acid, but for exact work this reagent is faulty as it does not stain all the fatty cells black.

Fatty Infiltration.—Fatty infiltration is likewise a retrogressive change in cell-life, but it has been separated (somewhat artificially) from fatty degeneration; it has been regarded as a simple accumulation of fat globules in a tissue and not as a change of the tissue elements themselves into fat; it is well seen in the liver; and it is due to an increased absorption or a diminished elimination of fat; it may be associated with fatty degeneration.

Fatuity.—Mental imbecility or stupidity; dementia; *Fatuitas Alpicolarum* or *Convallina* are synonyms of *Cretinism* (*q.v.*).

Fauces. *See* Diphtheria (*Clinical History, Faucial Diphtheria*); Malingering (*Digestive System, Faucial Ulceration from use of nitric acid*); Palate (*Anatomy*); Pharynx, Examination of; Pharynx, Diseases of; Physiology, Food and Digestion (*Structure of Alimentary Canal*).

Favus.

See also Nails, Affections of the (*Parasitic Diseases, Favus*); X-Rays (*Röntgen Ray Therapeutics, Skin Diseases*).

Favus (from favus, a honeycomb), also known popularly as "honeycomb" ringworm, is a disease of the skin caused by the growth on it of a fungus, the *Achorion Schönleinii.*

It affects especially the hairy scalp, but may occur on any part of the cutaneous surface, and in one case (Kaposi) it attacked the mucous membrane of the stomach and intestines and led to death.

The disease is curiously capricious in its geographical distribution, being common in Scotland, France, Russia, and Poland, while it is comparatively rare in England, Germany, America, Austria, and Italy. Subsequent to the expulsion of the Jews from Russia a number of cases were noted in London, but it does not appear to have spread to any extent to the local inhabitants.

Its first evidence is usually a variable amount of scaling on the surface, or if it occurs on the non-hairy skin this scaliness may be replaced by a moist dermatitis, which may assume a rounded form closely resembling ringworm. Exceptionally this eruption may be pustular.

After two or three weeks the typical cups or *scutula* may make their appearance. These are noted at first as little dull yellow points beneath the superficial layers of the epidermis, and are often pierced in the centre by a hair, for obviously the facilities for quiet uninterrupted growth are greatest in the hair follicles. The scutulum gradually enlarges, the epidermis over it gives way, and the typical sulphur yellow colour becomes more and more apparent. The scutula usually reach a size of from $\frac{1}{6}$ to $\frac{1}{4}$ of an inch, and in cases of the ordinary type of severity they press on adjacent ones, their edges are flattened, and the resemblance to a honeycomb becomes very evident.

If the case is neglected, the scalp is compressed between the accumulated scutula and the bony cranium, with the result that the hair-roots are permanently destroyed and the skin undergoes a species of cicatricial atrophy. If, on the other hand, the head is kept free of crusts, the growth of the hair is not interfered with and no baldness results. The affected hair has, however, a dull, lustreless appearance, and is often appreciably lighter in colour than the healthy hair in the neighbourhood.

Etiology. — The fungus which causes the disease was first demonstrated by Schönlein of Berlin, who in 1839 published a very brief note with a small drawing of the fungus, and subsequently in honour of his discovery the fungus was named the Achorion Schönleinii. The presence of the fungus in the crusts was soon recognised by observers elsewhere, and Gruby of Vienna (1841) and Hughes Bennett of Edinburgh (1842) confirmed the discovery and published excellent accounts of the fungus, Bennett's plates being exceedingly accurate, and his account of the seat of the scutulum entirely correct. He was, however, curiously loath to recognise the fungus as the cause of the disease, which he regarded as a "manifestation of tubercle." Both he and Gruby carried out numerous experimental inoculations, the latter on plants, reptiles, birds, and human subjects, with one successful inoculation—on a plant. Bennett's experiments were on human subjects—his patients, his students, and himself—and were all unsuccessful.

Recent experiments have, however, been more successful, and have demonstrated beyond all doubt that the fungus is the cause of the disease. As an example, we may take that of Winklereid Williams, who inoculated himself on the leg with pure cultures of the fungus, care being taken to irritate the skin and to keep the fungus in contact with the irritated surface. In three days signs of inflammation appeared; on the fourth there was an eruption of vesicles. After the ninth day minute yellow points began to appear, most, but not all of them, in relation to hair follicles, and these gradually grew into typical scutula. The disease is found in several of the lower animals, and it is probably from the mouse, through the cat, that the disease is conveyed to children. In mice the pressure effects of the scutula are very great, and lead to erosion of the bones of the cranium and to a fatal termination. The disease also occurs in hens and pigeons, but only exceptionally in horses and cattle, which are so frequently attacked by ringworm.

Microscopic Examination. — It usually suffices to mount a portion of the scutulum in a drop of liq. potass. After it has stood for some time the cover should be pressed on the slide so as to spread the material into a thin layer. A portion of scutulum examined in this way is found to consist almost entirely of fungus. Long filaments form a species of felt-work, among which the shorter portions commonly described as "spores" are evident.

When, on the other hand, a section of a scutulum is examined, while the margin is found to consist mainly of the long filaments, in the centre the shorter elements or "spores" are more numerous. Pressing down of the cover-glass on the ordinary specimen leads to separation of the individual elements, and such preparations show a misleading number of spores. The affected hairs contain long tubes of fungus, which usually completely obliterate all the internal structure of the hair, and their outsides are often sheathed with a felt-work of fungus, in which the joints are shorter than those inside.

The fungus may be stained by a number of methods, the best of which is Morris's modification of Gram's method. If it is desired to stain it in the hair, the hair should first be well washed in ether to remove all grease. The ether should not be allowed to evaporate, but should be washed off by alcohol and spirit, as otherwise it leaves a thin coating of fat on the surface of the hair, through which the stains do not easily penetrate. The stain used is the ordinary saturated solution of gentian violet in anilin water, and in this the hairs must remain at least half an hour. They are then placed into Gram's solution of iodine (iod. 1, pot. iod. 2, water 300 parts), in which they remain for three minutes. They should then be placed on a slide, firmly dried with blotting-paper, and a drop of pure anilin, containing enough iodine to give the solution a dark mahogany colour, placed over them. After washing in this for a few minutes the drop is washed off with pure anilin, which is in its turn washed off by xylol, and the specimen is then mounted in Canada balsam. Any fungus which is adherent to the outside of the hair is brilliantly stained, but the staining of the fungus within the hair is usually very irregular. The explanation of this probably is that the outside of the hair is so little diseased that it is only with difficulty that any of the stain makes its way into the interior of the hair. Sections of scutula stain perfectly with this method.

The amount of investigation to which the fungus of the disease has recently been subjected has led to the recognition of several varieties, for descriptions of which the works of Quincke and Neebe must be consulted. As yet their recognition has proved of little practical value.

Diagnosis.—In a well-marked case this is very easy. In no other disease are regularly shaped structures like the scutula produced, and consequently if they are present the nature of the case admits of no doubt. When they are not present in this typical form, the dry crumbling character and the bright sulphur yellow colour of the scales are very characteristic. The mousy odour which is so familiar in connection with the disease is due to the decomposition of dead fungus, and a somewhat similar odour is often noted in other diseases when the excretions are decomposing. Any doubt is set at rest by the examination of a portion of the crust under the microscope. If, as in any well-cared-for case, no crusts or scales are present, the diagnosis must be made from the examination of the hairs, when the long tubes of the fungus will be recognised in their interiors. Without this examination there might be some confusion with seborrhœa, but it will only be in very rare instances that the old plan of leaving a case untreated for a time to watch for the development of scutula is required.

Prognosis.—The prognosis of an untreated case is as bad as it is possible to be. There is absolutely no prospect, as there is in ringworm, of the disease dying out, and it will last until every hair on the scalp has been destroyed. In one case which came under the notice of the writer the disease had lasted no less than fifty years. In those countries where it is prevalent the disease thus becomes a matter deserving of the serious consideration of the civil authorities. Children with favus are rightly refused admission to any school, and belonging as they usually do to the lowest classes, they grow up altogether without education, and undoubtedly tend to drift into the criminal ranks. It is clearly the duty of the authorities to take the matter in hand and to take steps to stamp out the disease. Though not dangerous to life it is more dangerous to society than many diseases which are. The presently existing hospitals and dispensaries are quite unable adequately to cope with the disease; any treatment requires either months of treatment or the use of an apparatus expensive in the first instance and requiring skilled knowledge to work it. It is hardly too much to say that a case occurring in the lower classes, and only irregularly attending some institution, is to all intents and purposes incurable.

Treatment.—The treatment of any disease where the cause is so definitely known as in favus is exceedingly easy in theory. All that has to be done is to destroy the parasite and the thing is done. Unfortunately we are unacquainted with any remedy which will cause the destruction of the fungus in the depths of the follicles without at the same time destroying the skin. Antiseptics of various kinds are therefore of only limited value; they prevent the spread of the disease on the surface, but they do not cure the disease. Of the many in general use perhaps the first place should be accorded to the salts of copper and mercury. A good cheap ointment can be made by mixing together one drachm of sulphate of copper and one ounce of lard. The oleate of copper is rather more efficacious and much more expensive. Of the mercury salts probably the

best is the old-fashioned white precipitate ointment, which may be used in its full pharmacopœic strength. The ointment of the nitrate should be used diluted, commencing say with two drachms of the ointment to an ounce of vaseline, until experience has shown the amount of irritation which each patient's skin will stand. All ointment must be rubbed vigorously in at least twice daily, and the scalp should be kept covered with a cloth thickly spread with a somewhat weaker preparation. Their efficacy can of course be increased by covering this dressing with some impervious material, but if this is done the case must be carefully watched for signs of undue irritation, and this is not an easy matter in the class of patients which favus most affects. Another method of treatment aims at setting up so much reactionary inflammation, *e.g.* by blistering, that the fungus is indirectly destroyed. This method is suitable enough for cases where the disease is not extensive, but is quite unsuitable for those where practically the whole scalp is affected.

The only method of treatment which affords any reasonable hope of curing the disease is that of removal of all the diseased hair. The barbarous pitch cap or "calotte" is no longer used, but it at least proceeded on the right principle. Pitch plaster was applied to the shaven scalp, and when after some days the newly growing hair had become firmly adherent to it, it was forcibly removed, bringing the hair and a considerable portion of the scalp with it. Epilation by forceps was, up to very recently, our only practical method of dealing with the disease, and in careful hands and with a great deal of patience on both sides a cure might be hoped for in from six to twelve months, according to the extent of the spread of the disease. Unfortunately this period is too long for internal treatment in ordinary hospital wards, and too often the patient passed out of observation just as a mastery was being gained over the disease.

In the "X" rays we have at last obtained a means of epilation which promises to place the disease more completely in our power. Their action in causing the loss of hair has all along been known, but curiously it appears to have been only this year that it occurred to several dermatologists independently that here we had at last the remedy which we had so long sought for. By a judicious use of the X-rays the whole of the hair can be caused to fall from the scalp, and any fungus which remains is easily got at and easily destroyed. The treatment is still in its infancy, and it is consequently premature to be dogmatic on the method of application. We are at present using in the Edinburgh Royal Infirmary a coil which will carry a ten-inch spark, with one accumulator giving a current of six volts. Treatment is commenced with the tube twelve inches from the patient, and the exposure lasts for two minutes. If there are no signs of irritation the distance is shortened and the exposure lengthened, but this must be done gradually and carefully, as cases are extremely irregular in their response. (*See* "X-Rays.") For a varying number of weeks the patient has neither favus nor hair, but the latter gradually returns and the case is cured. The only disadvantages of the method are those due to want of experience in the use of the rays; if the exposure has been too prolonged or the current too strong the loss of hair may be permanent, though that is much to be preferred to favus, or, a more serious accident, there may be considerable sloughing of the skin, which not only leads also to the total and permanent loss of hair, but to an ulcer which is extraordinarily slow in healing. At the experimental stages of the treatment it is of course necessary to run as few risks as possible, and as experience grows the results will doubtless be more rapid; at present a month or six weeks is usually required to produce complete baldness, and perhaps two or three months more for the re-growth of the hair.

Favus of Non-hairy Skin.—When the disease attacks the non-hairy skin it is easily cured by the *constant* application of almost any antiseptic ointment or plaster, the mercury and carbolic plaster made by Beiersdorf of Hamburg being a most efficient and cleanly application.

Favus of the Nails.—This occurs in two forms. A scutulum may develop on the nail bed, and raise the nail plate over it, or the fungus may attack the substance of the nail and lead to its disintegration.

Both forms are difficult to manage. In the latter it is sometimes possible by scraping away the nail substance with broken glass and applying some antiseptic to cure the disease, but the simplest and the most radical method of treatment is avulsion.

Features. *See* PHYSIOGNOMY AND EXPRESSION.

Febricula. *See* EPHEMERAL FEVER; TROPICS, THE UNCLASSED FEVERS OF (*Continued Fevers of Short Duration*); UNDULANT FEVER (*Diagnosis*).

Febrifuges. *See* ANTIPYRETICS AND ANTIPYRETIC MEASURES; PHARMACOLOGY; TEMPERATURE; etc.

Febrile.—Relating to or accompanied by an elevation of temperature; feverish. *See* NEPHRITIS (*Etiology, Mild Cases, Febrile Albuminuria*); PURPURA (*Febrile Purpura*); TEMPERATURE.

Febriline.—A proprietary preparation said to consist of quinidine dissolved in lemon syrup.

Fecundation.—Impregnation; conception; fertilisation; the union of the male and female sexual elements, with, as the result thereof, the development of a new organism. *See* FŒTUS AND OVUM, DEVELOPMENT OF (*Fertilisation of Ovum*); PHYSIOLOGY, REPRODUCTION (*Impregnation*); SCROTUM AND TESTICLES, DISEASES OF (*Impotence, Sterility*); STERILITY.

Fecundity.—Productiveness; fertility; prolificacy; the capacity of bearing young; the probable fecundity of any marriage will depend upon a number of factors, including the age of the parents (especially of the mother) at marriage, the health of the parents (as regards syphilis, alcoholism, etc.), the use of checks to conception, heredity, the presence or absence of malformations or diseases of the genital organs, etc. The average fecundity of a marriage in Great Britain is about 4·5 births. In the lower animals feeding would appear to have a marked effect. Twinning and fecundity would seem to be related, and Jewish women are said to be very prolific. A case is referred to in the *Journal des sciences médicales de Lille* (Jan. 26, 1901) in which a woman is said to have given birth to 62 infants (41 female and 21 male) in 26 years, including twins (many times), quadruplets, quintuplets, and sextuplets (!). *See* VITAL STATISTICS (*Marriages, Births*).

Feeble-Mindedness. *See* MENTAL DEFICIENCY.

Feeding-Bottle. *See* INFANT FEEDING (*Artificial, Feeding-bottles*); INVALID FEEDING (*Feeding of Helpless Patients*); NURSERY HYGIENE (*Milk Supply, Feeding Bottles*).

Feeding, Forcible.—The feeding of those who refuse food (*e.g.* in melancholia); it is usually accomplished by the spoon, the nasal tube, or (best) by the stomach tube. *See* INSANITY, GENERAL TREATMENT OF (*Diet*).

Fees. *See* MEDICINE, FORENSIC (*Fees of Witnesses*).

Feet. *See* AINHUM (*Etiology, Toe-Rings, Chaps*); ALCOHOL (*Use of, in Tender Feet*); DEFORMITIES (*Lower Extremity, Toes, Foot*); ECZEMA (*Feet*); GOUT (*Irregular, Tender Feet*); SKIN, DISEASES OF SWEAT AND SEBACEOUS GLANDS (*Bromidrosis*); SKIN DISEASES OF THE TROPICS (*Pani Ghao, or Sore Feet of Coolies*); STOMACH AND DUODENUM, DISEASES OF (*Special Symptomatology, Cold Feet*); TABES DORSALIS (*Symptomatology, Feeling of Wool on the Soles of the Feet*); TOXICOLOGY (*Arsenic, Chronic, Keratosis of Feet*).

Fehling's Test. *See* URINE, PATHOLOGICAL CHANGES IN (*Sugar, Tests*); PHYSIOLOGY, FOOD AND DIGESTION (*Carbohydrates, Reduction of Metallic Salt*); GLYCOSURIA (*Detection, Fehling's Test*).

Feigned Eruptions. *See* DERMATITIS TRAUMATICA ET VENENATA (*Feigned Eruptions*); MALINGERING (*Varieties, Cutaneous*).

Fel Bovinum Purificatum.—Ox gall or bile, purified by washing with alcohol, and evaporating down to the consistence of a soft resin; it is used in doses of 5 to 15 grains, in cases of constipation (with pale stools) as a hepatic stimulant and purgative; it contains sodium taurocholate ($NaC_{26}H_{44}NO_7S$) and sodium glycocholate ($NaC_{26}H_{42}NO_6$).

Fellmongering.—The preparation of skins for the leather dresser; regarded as an "offensive trade."

Felo-de-se.—A person who "deliberately puts an end to his own existence, or commits any unlawful malicious act, the consequence of which is his own death" (*Blackstone*); or, a case or trial in which a verdict of felo-de-se or suicide is given.

Femoral. *See* ANEURYSM (*Situation, Lower Limb*); ARTERIES, LIGATURE OF (*Femoral*); HERNIA (*Inguinal, Diagnosis*); HERNIA (*Femoral*); HERNIA (*Radical Cure, Femoral*); PREGNANCY, AFFECTIONS OF GENERATIVE ORGANS (*Abnormalities in Position of Gravid Uterus*); SPINAL CORD, MEDICAL (*General Symptomatology, Reflexes, Femoral*).

Femur. *See* AMPUTATIONS (*Lower Limb*); DEFORMITIES (*Coxa Vara*); FRACTURES (*Femur*); HIP-JOINT, INJURIES OF (*Dislocation of Hip, Fractures of Femur, Coxa Vara*); KNEE-JOINT, DISEASES OF (*Sarcoma of Femur, Genu Recurvatum*); KNEE-JOINT, INJURIES OF (*Fractures, Separation of Epiphysis*).

Fenestra. *See* PHYSIOLOGY, SENSES, HEARING (*Middle Ear*).

Fennel Fruit.—The dried fruit of an umbelliferous plant (*Fœniculum capillaceum*), with an aromatic taste and odour, containing a volatile oil, and used in medicine in virtue of that oil; there is an official preparation, the Aqua Fœniculi, given in doses of 1 to 2 fl. oz.

Feredschik. *See* BALNEOLOGY (*Turkey, Roumelia*).

Fermentation. *See* GLYCOSURIA (*Detection, Fermentation Test*); MICRO-ORGANISMS (*Yeasts, Fermentative Bacteria, Varieties of Fermentation*); URINE, PATHOLOGICAL CHANGES IN (*Sugars, Tests*).

Ferments. *See* ALCOHOL (*Effect on Ferments*); ASEPTIC TREATMENT OF WOUNDS ("*Ferments" of Putrefaction*); ENZYMES.

Ferratin. *See* CHLOROSIS (*Treatment, Special*); FERRUM.—A non-official and easily digested preparation of iron, given in doses of from 8 to 15 grains.

Ferro-hæmol.—*See* Chlorosis (*Treatment, Special*).—A form of iron in organic combination.

Ferrometer. — An instrument devised by Jolles for estimating the amount of iron in the blood. An oxide of iron is obtained by reducing a measured quantity of blood (0·05 c.c.) to ashes in a platinum dish, adding acid sulphate of potash, and evaporating the combination to dryness. The residue is then dissolved in hot water, and dilute hydrochloric acid and ammonium sulphocyanide solution are added to give a red solution of sulphocyanide of iron. This solution is compared colorimetrically with a standard solution containing a known quantity of iron. The result does not, as was formerly supposed, give an accurate indication of the amount of hæmoglobin in the blood.

Ferro-Proteids. — Proteids linked to an iron-containing molecule, as in the pigment of the blood. *See* Physiology, Protoplasm (*Proteids*).

Ferropyrine. — A compound of perchloride of iron and antipyrine, which acts as a hæmostatic.

Ferrum. *See also* Anæmia; Children, Clinical Examination (*Examination of Fæces*); Chlorosis; Diet (*Mineral Constituents of Food*); Drug Eruptions (*Papular*); Pharmacology; Pigments of the Body and Excreta (*Melanins*); Toxicology (*Perchloride of Iron*); Urine, Pathological Changes in (*Inorganic Constituents, Metals*).

Metallic Iron has but one *official preparation*—Vinum Ferri—made by dissolving iron wire in sherry. It is acid in reaction, and can be prescribed along with arsenical preparations. *Dose*—1-4 ʒ. *Ferrum Redactum* is a powder containing about 75 per cent of metallic iron, along with some iron oxide. *Dose*—1-5 grs. *Preparation*—Trochiscus Ferri Redacti, 1 gr. in each.

The *salts of iron* may be arranged in two groups, according as they are derived from the lower oxide, FeO, or from the higher oxide Fe_2O_3. The former, the ferrous salts, are milder in action and generally more suitable for internal administration. The latter, the ferric salts, have more powerful local actions, and, although more stable outside the body, are, when taken internally, converted into the ferrous form before being absorbed. The official *Ferrous Salts* are the following:—1. *Ferri Sulphas*, translucent pale green rods, soluble 1 in $1\frac{1}{2}$ of water. *Dose*—1-5 grs. *Preparation* — Mistura Ferri Composita, "Griffith's Mixture," the iron being in the form of the carbonate. *Dose*—$\frac{1}{2}$-1ʒ. 2. *Ferri Sulphas Exsiccatus*, a white powder made from the sulphate by driving off most of the water of crystallisation by heat. *Dose*—1-3 grs. *Preparations*—(1) Pilula Ferri. Blaud's Pill. Made with sodium carbonate, which combines with the iron sulphate to form carbonate of iron. Each pill contains 1 gr. of ferrous carbonate. *Dose*—5-15 grs. (2) Pilula Aloes et Ferri. *Dose*—4-8 grs. 3. *Ferri Carbonas Saccharatus*, a mild preparation with no astringent action, well tolerated in large doses. *Dose*—10-30 grs., preferably in cachet. 4. *Ferri Arsenas*, a pale green powder when fresh, becoming blackened with time. It contains so little iron that its action is almost entirely that of arsenic. *Dose*—$\frac{1}{16}$-$\frac{1}{4}$ gr. 5. *Ferri Phosphas*, a pale blue powder. A mild drug, useful in rickets, scrofula, etc. *Dose*—5-10 grs. *Preparations*—(1) Syrupus Ferri Phosphatis, each fl. dr. containing 1 gr. of iron phosphate. *Dose*—$\frac{1}{2}$-1 ʒ. (2) Syrupus Ferri Phosphatis cum Quinina et Strychnina. Easton's Syrup. Each fl. dr. contains 1 gr. ferrous phosphate, $\frac{4}{5}$ gr. quinine sulphate, $\frac{1}{32}$ gr. strychnine. *Dose*—$\frac{1}{2}$-1 ʒ. 6. *Syrupus Ferri Iodidi.* The iodide of iron is made by heating together iron wire and iodine. *Dose*—30-60 m. Besides the above official forms, many other ferrous preparations, proprietary and otherwise, are in common use, among which may be mentioned Syrupus Ferri Phosphatis Compositus (Chemical Food, Parrish's Syrup); mixtures analogous to Easton's Syrup, made up in glycerin or malt; Easton's Syrup equivalents in tabloid and pill; and numerous Blaud's Pill substitutes in capsule and cachet. The official *Ferric Salts* are the following:—1. *Liquor Ferri Perchloridi Fortis*, a thick, dark, syrupy liquid, with strong local astringent action; not given internally. *Preparations*—(1) Liquor Ferri Perchloridi, 1 part of the strong solution to 3 of water, "Steel Drops." *Dose*—5-15 m. (2) Tinctura Ferri Perchloridi, same strength as the liquor. *Dose*—5-15 m. 2. *Liquor Ferri Persulphatis*, a dark red strongly astringent liquid. 3. *Liquor Ferri Pernitratis*, a clear brownish liquid, also astringent. *Dose*—5-15 m. 4. *Liquor Ferri Acetatis*, a deep red fluid. *Dose*—5-15 m. From *Ferri Peroxidum Hydratum* the following *scale* preparations are made:—1. *Ferrum Tartaratum*, dark red in colour and soluble in water. *Dose*—5-10 grs. 2. *Ferri et Quininæ Citras*, greenish scales, soluble 2 in 1 of water. 3. *Ferri et Ammonii Citras*, red scales, very soluble in water. *Dose*—5-10 grs. *Preparation*—Vinum Ferri Citratis. *Dose*—1-4 ʒ. Attempts have been made to procure a satisfactory organic compound of iron, but without much success; nor is there any sound reason why such a preparation should supersede the ordinary iron salts, suitably administered. "Carniferrin" and "Ferratin" are mixtures of meat and iron, given in doses of 8-15 grs. *Liquor Ferri Albuminati* and *Liquor Ferri Peptonati* are made with egg albumin. *Dose*—1-4 ʒ. The latter is a suitable preparation for administration to premature infants. *Liquor Ferri Dialysatus* (B.P. 1885) has now to a large extent dropped out of use.

The persalts of iron have a strong astringent action on ulcerated or bleeding skin surfaces or on mucous membranes. They are also efficient hæmostatics, and may arrest even severe hæmorrhage. The liquor ferri perchloridi is the most commonly used, applied directly on lint or cotton-wool, to stop bleeding from the nose, from piles, from the uterus, etc. It may also be painted on the tonsils and pharynx for astringent purposes. The tinctura ferri perchloridi given as a rectal injection (ʒi. to Oi.) is useful in the treatment of thread-worms. Internally the liquor ferri perchloridi has been given with apparent benefit in hæmorrhage from the stomach and intestines. In the treatment of diarrhœa the astringent iron salts are of little value. There seems to be no doubt that the tinctura ferri perchloridi, in doses of 20-30 m. frequently repeated, is of help in cases of erysipelas, septic sore throat, diphtheria, etc., although its mode of action in these diseases is by no means clear. The humid peroxide of iron, freshly prepared by mixing ℥iij. of liquor ferri perchloridi with ℥i. of sodium carbonate, diluted with water, is an excellent antidote in arsenical poisoning. In the treatment of anæmias the salts of iron stand unrivalled. When the percentage of hæmoglobin and the number of red blood corpuscles is below normal, iron increases both in a way that can be accomplished by no other drug. In the treatment of chlorosis its action is practically that of a specific, and in secondary anæmia, due to Bright's disease, phthisis, and the like, it is of great value. In pernicious anæmia it is absolutely worthless, a fact that may sometimes serve as a diagnostic aid. It is of little use, too, in the anæmia accompanying leucocythæmia. The most suitable forms to employ are the Blaud formula in one shape or another, and the saccharated carbonate, which is best given in cachet so as to avoid the blackening effect on the teeth of all iron compounds. Almost any preparation is efficient if suitably prescribed; but one must make certain that a sufficient quantity of iron is being administered. Treatment should be commenced with the equivalent of about 1 gr. of iron thrice daily, and this should be rapidly increased until 10 grs. per diem are being taken. Constipation is above all things to be avoided, and most authorities recommend that a brisk purge should precede the commencement of the course of iron. In a great variety of conditions associated with debility, even where a blood examination reveals little or no anæmia, iron is given as a tonic, and for this purpose the scale preparations are specially recommended. In convalescence from acute disease Easton's syrup is frequently prescribed. In chronic heart disease the addition of iron to the heart tonic employed may prove beneficial. In chronic Bright's disease tinctura ferri perchloridi is largely used, and has a considerable reputation as a diuretic. It is probable, however, that its real value lies in counteracting the secondary anæmia usually present, and it is useless to prescribe it unless there is some anæmia. The syrup of the iodide is specially recommended for diseases associated with wasting or faulty nutrition, and it has also been used in rheumatoid arthritis.

Fersan.—A preparation (from the blood of the ox) said to consist of an organic compound of acid albumin with iron and phosphorus.

Fertilisation. *See* Fecundation; Fœtus and Ovum, Development; etc.

Fester. *See* Suppuration.

Festination.—The tendency to hurry (and flurry) in the doing of anything, which is noticeable in some forms of insanity and nervous diseases.

Fetus.—The unborn infant between the end of the second month and the full term of intra-uterine life; the young of the lower animals. The spelling *fetus* is etymologically correct, but the form *fœtus* is still in common use save in the *British Medical Journal* and American medical works. *See* Fœtus.

Fever. *See* Actinomycosis; Anthrax; Appendicitis; Balneology (*Actions of Baths, "Critical Fever"*); Beriberi; Blackwater Fever; Cholera; Dengue; Diphtheria; Dysentery; Ephemeral Fever; Epidemiology; Erysipelas; Filariasis (*Elephantoid Fever*); Foot-and-Mouth Disease; Fourth Disease; Gall-Bladder and Bile Ducts Diseases (*Inflammatory*); Glanders; Glandular Fever; Gonorrhœa; Hæmoglobinuria (*in Fevers*); Hæmorrhage (*Hæmorrhagic Fever*); Heart, Myocardium and Endocardium (*Malignant Endocarditis*); Hydrophobia; Hysteria (*Disorders of General Nutrition, Hysterical Fever*); Infection; Inflammation; Influenza; Invalid Feeding (*in Pyrexial States*); Joints, Diseases of (*Clinical Features, Tuberculous Arthritic Fever*); Kidney, Surgical Affections (*Pyelitis, etc.*); Leprosy; Leucocythæmia (*Acute*); Leucocytosis; Liver, Diseases (*Abscess*); Lung, Tuberculosis of (*Pyrexia*); Lymphatic System (*Physiology and Pathology, Hodgkin's Disease*); Malaria; Mumps; Measles; Plague; Puerperium, Pathology (*Fever*); Puerperium, Physiology (*Temperature, Milk Fever*); Pyæmia; Relapsing Fever; Rheumatism, Acute; Rubella; Scarlet Fever; Septicæmia; Syphilis; Temperature; Tetanus; Tropics, Unclassed Fevers of; Trypanosomiasis; Tuberculosis; Typhoid Fever; Typhus Fever; Undulant Fever; Urine, Pathological Changes in (*Significance of Albuminuria*); Varicella; Whooping-Cough; Wounds (*Traumatic Fever*); Yaws; Yellow Fever.

Fevers.—Among the many diseases to which the term *Fever* has been applied, the following may be specially named:—Abdominal Fever (Typhoid), Acclimatisation Fever, Adynamic Fever (Typhoid or Typhus), African Fever (Malaria), Algid Fever, Ambulatory Type of Fever (patient not confined to bed), Anginous Fever, Ardent Fever (Thermic, etc.), Arthritic Fever, Asthenic Fever, Atrabiliary Fever (Hæmorrhagic Malaria), Autumnal Fever (Typhoid, Malaria, etc.), Bastard Fever (Anomalous), Bengal Fever (Remittent), Biliary Fever, Bilious Fever, Blackwater Fever, Bouquet Fever (Dengue), Brain Fever, Breakbone Fever (Dengue), Bulam Fever (Yellow Fever), Burdwan Fever (Typhomalarial), Catamenial Fever, Catarrhal Fever, Catheter Fever, Cerebro-Spinal Fever, Cesspool Fever (Typhoid), Childbed Fever (Sepsis), Choleraic Fever (Malaria), Congestive Fever, Continued Fever, Convulsive Fever, Crete Fever (Malaria), Cyprus Fever (Malaria), Dandy Fever (Dengue), Deccan Fever (Malaria), Dengue Fever, Diathetic Fever, Diphtheritic Fever, Dum-Dum Fever (Kala-azar or Piroplasmosis), Dysenteric Fever, Emotional Fever, Enteric Fever (Typhoid), Ephemeral Fever, Epidemic Fever (Various), Erratic Fever, Eruptive Fever (Various), Erysipelatous Fever, Essential Fever, Exanthematic Fever, Famine Fever (Relapsing), Fatigue Fever, Fifteen Day Fever (Relapsing), Five Day Fever (Relapsing), Ganglionic Fever (Glandular), Gaol Fever (Typhus), Gastric Fever (Various), Gibraltar Fever (Yellow Fever?), Glandular Fever, Gouty Fever, Growing Fever (Febricula, Rheumatism?), Hæmaturic Fever, Hæmorrhagic Fever (Various), Harvest Fever, Hay Fever, Heat Fever, Hectic Fever, Hepatic Fever, Herpetic Fever, Hill Fever (Malaria), Hospital Fever (Typhus), Hydrophobic Fever, Hysterical Fever, Icteric Fever, Idiopathic Fever, Infantile Fever (Various), Infectious Fever (Various), Inflammatory Fever, Intermittent Fever (Malaria), Jail Fever (Typhus), Jungle Fever (Malaria), Latent Fever (mild form of Scarlet, Typhoid, etc.), Leghorn Fever (Yellow Fever), Levant Fever (Malaria), Lochial Fever, Low Fever (Asthenic or Adynamic), Lying-in Fever (Puerperal Fever), Macular Fever (Typhus), Malarial Fever (Various), Malignant Fever (Various), Malta Fever (Undulant Fever), Marsh Fever (Malaria), Masked Fever (Various), Mediterranean Fever (Undulant Fever), Mesenteric Fever (Typhoid), Miliary Fever, Milk Fever, Mysore Fever (Malaria), Neapolitan Fever (Typhomalarial), Nephritic Fever, Nervous Fever, Niger Fever (Malaria), Octan Fever (Malaria), Oyster Fever (Typhoid), Paludal Fever (Malaria), Paroxysmal Fever (Various), Periodical Fever, Pernicious Fever (Various), Peshawur Fever (Typhomalarial), Petechial Fever (Typhus, Epidemic Cerebro-spinal Meningitis), Pharyngeal Fever, Phthisical Fever (Hectic), Pleuritic Fever, Pleuro-pneumonic Fever, Pneumonic Fever, Puerperal Fever (Various), Purpuric Fever, Putrid Fever (Typhus, Typhoid), Pyæmic Fever, Pyogenic Fever, Quartan Fever (Intermittent), Quinine Fever, Quintan Fever (Intermittent), Quotidian Fever (Intermittent), Recurrent Fever, Relapsing Fever (Famine Fever), Remittent Fever (Malaria), Rheumatic Fever, Rock Fever (Gibraltar), Rose Fever (Hay Fever, Erysipelas in Scotland), Sapræmic Fever (Puerperal), Scarlet Fever, Scinde Fever (Malarial or Typhomalarial), Scorbutic Fever, Semiterrian Fever (Malaria), Septan Fever (Intermittent), Septicæmic Fever, Septic Fever, Sextan Fever (Intermittent), Ship Fever (Typhus), Siam Fever (Yellow Fever), Simple Fever (Febricula), Slow Fever, Smyrna Fever (Malaria), Spirillum Fever (Relapsing), Splenic Fever (Anthrax), Spotted Fever (Typhus, Epidemic Cerebro-spinal Meningitis), Spring Fever, Starvation Fever, Sthenic Fever, Stiff-necked Fever (Dengue), Summer Fever (Hay Fever), Sun Fever (Sunstroke or Dengue), Surgical Fever, Sweating Fever, Swine Fever, Symptomatic Fever, Syphilitic Fever, Tertian Fever (Intermittent), Thermic Fever (Sunstroke), Toxæmic Fever, Traumatic Fever, Tropical Fever (Yellow Fever and Various), Tuberculous Fever, Typhoid Fever, Typhomalarial Fever, Typhus Fever, Uræmic Fever, Urethral Fever, Vaccinal Fever, Varicellar Fever, Varioloid Fever, Variolous Fever (Small-pox), Verminous Fever (Worm Fever), Vesicular Fever (Pemphigus), Yellow Fever.

Fibres. *See* PHYSIOLOGY, TISSUES (*Elastic, Muscular, Nervous, etc.*).

Fibrin.—A proteid, a globulin, of the blood, coagulated by heat; fibrinogen is the globulin in its soluble state. *See* BLOOD, PHYSIOLOGY OF (*Coagulation*); ENZYMES (*Fibrin Ferment*); PHYSIOLOGY, BLOOD (*Coagulation*).

Fibrinogen. *See* FIBRIN.

Fibrinous. *See* BRONCHI, BRONCHITIS (*Acute, Morbid Anatomy, Fibrinous Type*); NOSE, ACUTE INFLAMMATION (*Fibrinous Rhinitis*).

Fibrinuria. *See* URINE, PATHOLOGICAL CHANGES IN (*Chyluria, Hœmaturia*).

Fibro-Adenoma. *See* MAMMARY GLAND, DISEASES OF (*Neoplasms*).

Fibroblasts.—Mucoid tissue cells which are transformed into fibres. *See* PHYSIOLOGY, TISSUES (*Fibrous*).

Fibro-cartilage. *See* PHYSIOLOGY, TISSUES (*Cartilage, Elastic, White*).

Fibro-chondroma.—Branchial fibro-chondromata or cervical auricles are small congenital growths, which are regarded as developed in connection with the third or fourth branchial cleft.

Fibroid. *See* Curettage, Uterine (*Uses, Hæmorrhage from Uterine Fibroids*); Electricity (*Galvanism, Gynecology, Electrolysis of Fibroids*); Heart, Myocardium and Endocardium (*Special Pathology, Affections of Myocardium, Fibroid Disease*); Labour, Prolonged (*Faults in the Soft Passages, Uterine Fibroids*); Lungs, Pulmonary Fibrosis (*Fibroid Phthisis*); Pregnancy, Diagnosis (*Differential, Uterine Fibroids*); Skin, Tuberculosis of (*Lupus, Fibroid*); Stomach and Duodenum, Diseases of (*Fibrosis*); Uterus, Non-Malignant Tumours of (*Fibroids or Fibro-myomata*).

Fibroma. *See* Bone, Diseases of (*Tumours, Fibroma*); Brain, Tumours of (*Morbid Anatomy, Fibromata*); Neck, Region of (*Solid Tumours, Fibromata*); Orbit, Diseases of (*Solid Tumours, Fibromata*); Ovaries, Diseases of (*Fibroma*); Peritoneum, Tumours of (*Innocent, Fibromata*).

Fibromatosis. *See* Mammary Gland, Diseases of (*Hypertrophy, Diffuse Fibromatosis*).

Fibro-myoma. *See* Fibroid; Uterus, Non-Malignant Tumours of.

Fibrosis.—Arterio-capillary fibroid degeneration. *See* Arteries, Diseases of (*Arterio-sclerosis*)

Fibula. *See* Ankle-Joint, Region of, Injuries (*Pott's Fracture*); Deformities (*Bow-Legs, Curved Fibula*); Fractures (*Bones of Leg, Fibula*); Knee-Joint, Injuries of (*Fracture of Upper End of Fibula, Separation of Epiphysis*).

Ficus.—The dried receptacles of the urticaceous plant, *Ficus carica*; figs are used, sometimes in combination with stewed rhubarb, as a mild purgative in chronic constipation; they are used in making the official preparation *Confectio Sennæ*.

Ficus Unguium.—A disease of the nails in which there is recession of the skin at the root of the nail with exposure of the root. *See* Nails, Affections of.

Fidgets.—Marked recklessness and irritability, due to fatigue, dyspepsia, rheumatism, etc.; dysphoria.

Field of Vision. *See* Vision, Field of.

Fièvre de Croissance. *See* Bone, Diseases of (Growth-Fever, *Osteomyelitis*).

Fifth Nerve, Affections of.

See also Brain, Physiology of (*Cranial Nerves, Fifth*); Brain, Tumours of (*Localising Symptoms, Cranial Nerves*); Brain, Cerebellum, Affections of (*Tumours, Symptoms, Cranial Nerves*); Cornea (*Ulcerative Keratitis, Causes, Fifth Nerve Paralysis*); Nerves, Neuralgia (*Trigeminal*); Tabes Dorsalis (*Symptoms, Affections of Cranial Nerves*).

The central connections of the fifth nerve are referred to vol. i. p. 469. But it is necessary to describe briefly the course and functions of the three branches arising from the Gasserian ganglion.

The nerve contains motor and sensory fibres, and as the nuclei and the roots are distinct, lesions may be limited to sensory or motor nuclei, roots, or divisions, and may if sensory be limited to a part only of these fibres.

Anatomy (*see* Diagram on p. 269).—The sensory and larger root passes into the Gasserian ganglion which is formed on it, and which lies in the cavum Meckelii on the summit of the petrous part of the temporal bone; the smaller root which contains all the motor fibres passes downwards from the inner side of the large sensory root, and runs outwards below the ganglion to join the lowest of the three branches into which the larger root divides upon leaving the ganglion.

The first or *ophthalmic branch* is the highest of the three, and enters the orbit through the sphenoidal fissure in close relationship to the third and fourth nerves, and supplies branches to the dura mater, the eyeball and lachrymal gland, the mucous membrane of the nose and eyelids; to the skin of the nose, upper eyelid, the forehead and the upper part of the scalp, besides having many communications with the third and fourth nerves.

The second or *superior maxillary branch* passes through the foramen rotundum, crosses the spheno-maxillary fissure, and reaches the face by the infraorbital foramen. Its branches pass to the upper lip, lower eyelid, and side of the nose, cheek, and arterior part of the temple; the teeth in the upper jaw; the mucous membrane of the nose, of the upper part of the pharynx, of the antrum of Highmore, and the posterior ethmoidal cells; the soft palate, tonsils, and uvula; and with Meckel's ganglion supplies the glands and mucous structures in the roof of the mouth.

The third or *inferior maxillary branch* is the largest of the three, and is made up of a large sensory division from the Gasserian ganglion, together with the small motor root which is distinct from the ganglion. The two join after passing through the foramen ovale. The nerve

divides into two branches, of which the smaller anterior division contains the motor fibres which go to supply the masticatory muscles, and also the buccal branch which is sensory, and which supplies the outer and inner aspects of the cheek as far forward as the angle of the mouth.

The larger posterior division is mainly sensory, and supplies part of the skin on the side of the head and the region of the ear, including the external auditory meatus, the lower lip and lower part of the face, the greater part of the tongue and the lower teeth and gums, part of the mucous membrane of the mouth, the salivary glands, the articulation of the lower jaw, part of the dura mater and skull, and the lining membrane of the mastoid cells. Motor fibres also pass in this division to the mylohyoid, the anterior belly of the digastric, the tensor palati, and tensor tympani muscles.

As already indicated, the sensory fibres of the three branches of the trigeminus have a defined field of action, but also a wide area of skin, which may be supplied by one branch, or possibly several branches.

The region of the ear described as being supplied by the third branch may be mainly supplied by cervical nerves, and Zander has definitely proved that in the middle line of the face the skin is supplied from the nerves of both sides. The course of the taste fibres will be referred to later.

Physiology.—The physiology of the fifth nerve has already been indicated in the preceding paragraphs.

The motor fibres supply the muscles of mastication, the mylohyoid, the posterior belly of the digastric, and almost certainly the tensor palati and the tensor tympani muscles.

The sensory fibres supply the skin of the face, the eyeball, and eyelids, the nose, the forehead, part of the scalp, and the region of the ear. The mucous membrane of the nose, mouth, and gums, and the teeth in both jaws are also included in the domain of this nerve.

The trophic functions of the nerve are referred to in some detail under "Paralysis of Sensory Fibres," and it is only necessary to state here that while the healthy nutrition of skin, etc., depends on the integrity of the sensory and motor nerve-fibres, the cornea ulcerates and trophic ulcers occur in skin and mucous membrane as the result of an irritative and not a destructive lesion. The teeth certainly tend to loosen and decay when their nerve-supply is cut off, and therefore the fifth nerve is essential for their healthy existence; so that a destructive lesion of the superior maxillary or inferior dental nerves would, as far as they are concerned, be followed by quite as serious consequences as an irritative.

The tongue is supplied with common sensibility by the fifth nerve, while taste is supplied to the anterior two-thirds of the tongue by fibres reaching the lingual nerve by the chorda tympani and to the posterior one-third by the glosso-pharyngeal. It should be noted, however, that clinical evidence strongly supports the belief that all these taste fibres eventually enter the pons by the sensory root of the fifth nerve.

The nasal mucous membrane, the lachrymal apparatus, and the glandular structures in nose and mouth, are supplied by the fifth, therefore in lesions of the nerve the eyes may suffer from lack of tears, while smell and taste may be considerably interfered with, although the olfactory and gustatory nerve-fibres are unaffected.

Paralysis of the Nerve or its Branches

Etiology.—Paralysis of the fifth may be of central origin. Hæmorrhage, softening, and tumours of various kinds may damage the cortical centres, with regard to which we can say that we can at present locate the motor only, the fibres between cortex and nuclei, the nuclei, the ascending root; and the motor nuclei may be affected in chronic bulbar paralysis. If the lesion is confined to the pons, then the sensory and motor fibres of the nerve may be involved; while in diseases of the cord, in which the nerve may be affected, such as locomotor ataxia and syringomyelia, the sensory fibres alone suffer.

The nerve as a whole may be involved by meningeal inflammation, by tuberculous, syphilitic, or other tumours, and by caries of the temporal bone.

A primary neuritis is seldom met with affecting this nerve, and it has only occurred in a few isolated cases, which have been described as of rheumatic or gouty origin. The nerve very rarely suffers in alcoholic or other form of peripheral neuritis. But neuritis of the fifth may be secondary to many conditions, injuries, tumours, local inflammation, and the like, and may affect the nerve as a whole, or one or other of its branches.

While all the three branches may suffer from meningeal inflammation, tumours, or injuries in the middle fossa, and perhaps specially fracture of the base of the skull, the first branch is liable to damage from intraorbital tumours and so forth, and may be involved by an aneurysm springing from the internal carotid in the neighbourhood of the nerve, or from tumours of the pituitary body. All the branches may be injured by cuts or superficial lesions.

Clinical Features. — *Paralysis of Motor Fibres.*—If there is unilateral paralysis then mastication is weak on the affected side, and when the fingers are placed over the muscles, and the attempt is made to close the jaws, then the weak, flabby temporal and masseter muscles contrast sharply with the firmly-contracted muscles on the healthy side. The pterygoid muscles act on one side alone, and when the mouth is opened the lower jaw is

deflected towards the paralysed side, and lateral movements of the jaw can only be made towards and not away from the affected side.

Usually electrical changes occur in the affected muscles, sometimes including the reaction of degeneration, and eventually the muscles waste. This wasting causes flattening of the temporal and zygomatic fossæ, and muscular shortening may develop, limiting somewhat the extent to which the mouth can be opened. Remak describes subluxation of the lower jaw on the affected side, when the mouth is opened, but this has not been generally noted.

No definite phenomena indicating paralysis of the mylohyoid and posterior belly of the digastric muscles can be made out, unless it be that the floor of the mouth is less resistant on the affected side when tested with the finger (Oppenheim). Müller and Schmidt believe that the palatal arch droops on the paralysed side, but Gowers and the majority of neurologists doubt the existence of any definite manifestation of paralysis of the tensor palati, although they believe that the muscle derives its nerve-supply from the fifth. Similarly in no recorded case has any definite symptom resulting from paralysis of the tensor tympani been observed.

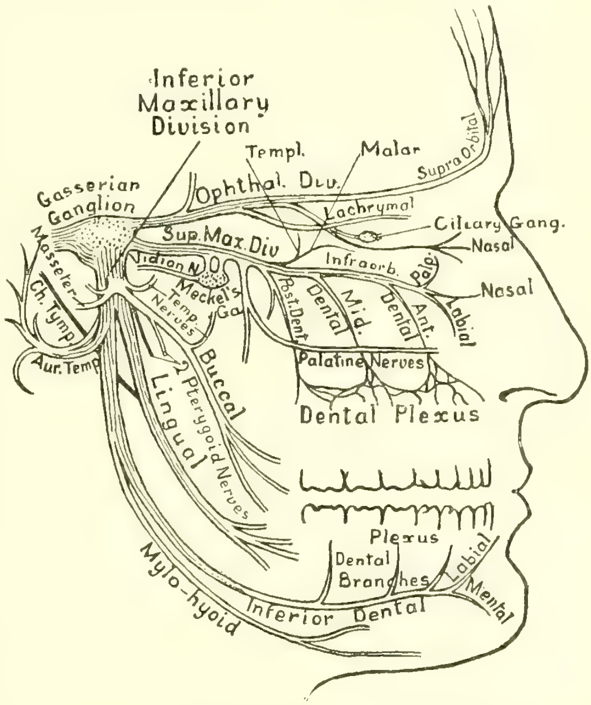
In cases where the paralysis is bilateral, the lower jaw cannot be firmly opposed to the upper.

Referring for a moment to the etiology of masticatory paralysis as an isolated lesion of the fifth, hæmorrhage, areas of softening and tumours in the pons have produced it, but there are few, if any, cases recorded in which a definite limited cortical or subcortical lesion has caused the condition. Turner mentions several cases recorded by Barlow, Dalmont, and Kirchoff, but in these the lesions and the paralysis resulting therefrom were very extensive. Masticatory paralysis is far more commonly dependent on a peripheral than on a central lesion.

Paralysis of the Sensory Fibres.—The sequence and the extent of the phenomena vary considerably, mainly dependent upon the nature of the lesion. If the lesion is of an irritating nature, or of slow onset, pain may be the first symptom, sharp or burning, and often referred to the distribution of the affected branch or branches of the nerve, with generally marked painful points along the line of the nerve.

The skin may be hyperæsthetic especially to pain during this stage, and this may last even after tactile sensibility has been diminished or lost (*see* "Nerves, Neuralgia").

Pain may, however, be absent and anæsthesia may be the first symptom, tactile sense first being affected. The muscles of expression become anæsthetic as well as the skin, so that grimaces may be voluntarily made without the individual being sensible of their performance. Contractions of facial muscles are said to be slightly delayed in execution, probably from loss of common sensation. The mucous membranes of the mouth, nose, and tongue are anæsthetic on the affected side, and a well-known phenomenon is the description given by the patient when drinking out of a cup, that he feels as if it were broken on the paralysed side, and not infrequently the anæsthetic mucous membrane of



Diagrammatic representation of the three divisions of the fifth nerve with their principal branches.

the cheek is bitten by the teeth, and the bitten part heals abnormally slowly, and may even tend to ulcerate. The patient does not chew with the affected half of his jaw because he is unable to locate the food, and food is apt to accumulate between the teeth and the cheek. The nasal mucous membrane becomes dry, and smell is in time interfered with, due possibly to trophic changes in the olfactory cells, possibly, however, as Krause and Majendi believe, because the fifth nerve has a definite share in the recognition of the sense of smell. Sneezing cannot be induced by stimulation of the affected nostril.

The ocular conjunctiva may be anæsthetic, and tears do not flow from the affected eye. Where this occurs it has been thought to depend on a lesion either at or peripheral to the spheno-palatine ganglion. The conjunctival reflex is also lost.

Special reference will be made to common sensibility of the tongue along with changes in taste sensation later, meanwhile it is only necessary to state that as the whole of the tongue, the palate, and fauces derive their common

sensibility from the fifth, there may be unilateral anæsthesia, sometimes complete, but not infrequently inexplicably limited to the anterior two-thirds of the tongue.

There are also marked trophic and vaso-motor disturbances due to affections of the sensory fibres of the trigeminus, some of which have given rise to much discussion. Not infrequently a herpetic eruption appears along the line of the supraorbital branch, probably due to a neuritis, and which may in exceptional cases be of great severity, causing intense pain and associated with iritis even and sometimes corneal ulceration. Perhaps an even commoner herpetic eruption found in the domain of the fifth nerve is that which occurs in the lips, herpes labialis, and which is so often associated with ordinary coryza and lobar pneumonia. Trophic phenomena vary according as the lesion is an irritative or destructive one, and the herpetic eruption as above described is a good example of the former.

Diminution of nasal and lachrymal secretions has been already referred to, and along with the salivary such diminution is the result of a destructive lesion, while a temporary excessive flow may follow an irritative. It is stated that the affected side of the face is often paler owing to diminished vascularity, but the writer has never observed this phenomenon.

Progressive neuro-paralytic ophthalmia, or a condition beginning with dryness of the cornea, then opacity, and later ulceration which may eventually cause perforation and lead to panophthalmitis, is the most disputed phenomenon met with in some affections of the first branch of the trigeminal nerve. It was thought at one time (Majendie) that section of the nerve would result in such trophic disturbance, but experimental evidence, now amply supported by undoubted clinical data, demonstrates that it is the result of irritation and not destruction of the ophthalmic division of the fifth, or possibly in some cases irritation of the Gasserian ganglion. It might be supposed that an insensitive cornea should be readily injured by foreign bodies, and aided by the diminished flow of the lachrymal secretion, but this is not demonstrated by clinical facts. Still, when as already mentioned the mucous membrane of the affected cheek is bitten accidentally, healing is slow and ulceration is not uncommon, it does seem as if the fifth was a trophic nerve. Meissner has suggested that the keratitis is due to section or possibly irritation of a definite bundle of trophic fibres which are situated near the inner side of the Gasserian ganglion, and then pass into the ophthalmic division of the fifth; but on the other hand, the numerous successful operations for relief of trigeminal neuralgia by removal of the Gasserian ganglion without the production of any progressive keratitis, and the experiments of Ferrier and Turner on monkeys, confute any such statement. The neuro-paralytic keratitis is only due to irritation, and probably only when the irritant is of a very virulent nature, such as might be produced in a septic wound exposing the nerve or ganglion, or where a tumour presses on the nerve. If this is true the condition must be the result of a neuritis (*see* "Cornea").

The Prognosis depends entirely on the nature of each case. It has been impossible to describe separately lesions which are purely irritative and those which are destructive.

Irritative phenomena are often succeeded by those which are destructive or paralytic, and the evidence of complete destruction of a branch or of the whole nerve is always ominous. Where the motor fibres are affected incomplete reaction of degeneration, and specially the continuance of reaction with the faradic current, are favourable signs.

Diagnosis. — The diagnosis of peripheral lesions which give definite irritative or paralytic phenomena is easy, and a knowledge of the course and functions of each of the three branches enables us to localise the position, and to guess at the nature of the disease. Anæsthesia limited to the area supplied by the first division, and especially if there coexists unilateral ocular paralysis, suggests a lesion either at the sphenoidal fissure or in the orbit.

Anæsthesia of the area supplied by the second division suggests a lesion at the spheno-maxillary fissure, or in the superior maxillary bone. A complete lesion of the third division rarely occurs alone. The second and third divisions may be affected without the first in disease at the spheno-maxillary fissure or in the sphenoid bone. Complete anæsthesia of all the areas supplied by the three divisions of the trigeminus, and without the motor root being implicated, would point to a lesion of the sensory root between the Gasserian ganglion and the pons, or possibly a very localised pontine lesion, supposing that there were no impairment of sensory or motor function in the limbs. The motor root is closely related to the sensory, so that it must be admitted that a lesion of the Gasserian ganglion, or of the sensory root between the ganglion and the pons, could hardly fail to implicate the motor fibres.

Passing backwards to the pons, the sixth nerve emerges at no great distance from the fifth, and complete paralysis of the two nerves definitely locates the lesion at the pons.

Where the fifth is paralysed on one side, and the arm and leg on the opposite side, the lesion is pontine and below the decussation of the fibres of the fifth, whereas if trigeminal anæsthesia occurs on the same side as anæsthesia of the limbs, the lesion may be in the upper part of the pons, the crura cerebri, or posterior part of the posterior limb of the internal capsule.

Gowers states "that the associated paralysis of the conjugate movements of both eyes towards the side of the lesion is conclusive evidence of disease within the pons."

In conclusion, pain due to an irritative lesion of the fifth or one of its branches is more persistent than a mere functional neuralgia can ever be, and the addition of anæsthesia may clear up a case otherwise doubtful.

Treatment.—Once the lesion has been localised, then the next step is to attempt its removal where this is possible.

Where syphilis is a probable cause, suitable and prompt treatment should be commenced. Pain should be soothed by fomentations and poultices, but the utmost care must be taken of the skin, which is easily damaged by too hot applications. Blistering or counter-irritation should be limited to skin not supplied by any branches of the affected nerve, and therefore such treatment is generally undesirable. Sedatives internally, such as opium, gelseminum, and chloral, and locally, such as morphia and cocaine, may be required.

Faradic or galvanic stimulation is often useful where the nerve-fibres are not affected beyond hope of recovery, or where recovery of power of conducting impulses is actually in progress. Strychnine hypodermically injected near the affected nerve has been found useful in a few cases.

When there is the smallest reason to suspect that there is progressive neuritis present, by which term inflammation in the nerve is implied, then absolute rest should be enjoined, and electricity and such remedies as strychnine must on no account be used.

Affections of Taste

It may not be out of place to remind the reader that the sense of taste is specially acute at the root of the tongue along the line of the circumvallate papillæ, in front of the anterior pillar of the fauces, along the edges, and at the tip of the tongue, and that the dorsum is a much less sensitive surface. Certain parts of the gustatory mucous membrane appear to be peculiarly adapted for the recognition of special qualities of taste, such as bitterness and sweetness which are most easily appreciated with the back of the tongue, and sourness and saltness which are better recognised at the tip and edges.

It must also be remembered that acuteness of taste varies in different individuals, and becomes dulled with the advance of old age. Sugar, salt, quinine, citric acid, but specially a weak galvanic current, are good methods of testing the sense of taste. The tongue should be protruded, and a little of the powder, or, better still, a fairly strong solution, rubbed upon the part of the organ to be tested. Recognition of the quality of the substance tasted should be indicated by signs or in writing. No substance should be used which stimulates nerve-endings subserving common sensation. If galvanism is used, two wire electrodes connected with one or at most two cells should be placed very close together on the tongue, when, if gustatory sensation is intact, a peculiar metallic taste is at once felt by the patient. This is an accurate method, and one of very easy application.

A weak current should invariably be used, because pain is inevitably experienced where a strong current is employed, and this must obscure an otherwise accurate test for taste sensation. One great advantage obtained by using a weak galvanic current is, that it alone gives an absolutely temporary sensation, whereas salt, sugar, etc. all leave for a considerable period of time a more or less lasting taste impression, thus greatly protracting the examination and even rendering accurate diagnosis difficult.

Perhaps an even better method of employing the galvanic current has been suggested by Neumann. It consists in placing the neutral electrode on the sternum, and the pointed diagnostic electrode is then applied to the tongue. With the positive pole or anode an acid sensation is experienced, while with the negative or cathode it is alkaline.

The tongue, the soft palate, the fauces, and the buccal surface of the epiglottis contain the peripheral taste organs. The anterior two-thirds of the tongue is supplied by taste fibres through the chorda tympani nerve, the posterior third by the glosso-pharyngeal, and the soft palate and fauces by branches from Meckel's ganglion. It seems probable, however, that all the taste fibres, for the tongue at least, pass eventually into the sensory root of the fifth.

The fibres for the anterior two-thirds of the tongue are distributed to that organ by the lingual branch of the fifth, from which they pass into the chorda tympani, thence to the seventh, thence they pass by the great superficial petrosal and vidian nerves to the sensory root of the fifth. An isolated case recorded by Ferguson, in which an exostosis pressed upon and destroyed the left vidian nerve, proves the course taken by these fibres with considerable definiteness. It produced both loss of taste in the left half of the anterior two-thirds of the tongue and descending degeneration in the great superficial petrosal nerve, which could be traced through the geniculate ganglion, thence into the facial, thence into the chorda tympani, and finally into the lingual nerves.

The fibres for the posterior third of the tongue, which are distributed by the glosso-pharyngeal nerve, probably leave that nerve by the nerve of Jacobson, and then by the small superficial petrosal nerve reach the otic ganglion, and thence pass into the third division of the fifth.

Erb, Schiff, Vulpian, Gowers, and others have

recorded cases and experiments which go to prove that lesions of the root of the fifth cause loss of taste, and especially is there strong evidence with regard to the anterior two-thirds of the tongue. But on the other hand it is difficult to explain many contradictory statements as to the branch or branches of the fifth which convey taste fibres. There is loss of taste with a lesion of the first and second divisions only, while with no loss of taste sensations there have been either (1) a lesion of the third division only, or (2) the first and second divisions only. So far, however, taste sensations are obviously carried by the second or third branch of the fifth, or perhaps both, and the taste fibres are unquestionably present in the root of the nerve, although it is probable that the glosso-pharyngeal fibres may pass into the roots of that nerve, or as stated above by Jacobson's nerve join the third division of the fifth. Once the taste fibres have reached the pons there is much doubt as to their future course, they certainly decussate, and they probably pass to the taste centre, believed to be situated at the tip of the temporo-sphenoidal lobe.

Loss of taste may occur as a part of hemianæsthesia in a lesion of the internal capsule or brain, but it is more complete and certainly more definitely limited when the lesion is one of the nerve or nerves conveying gustatory fibres.

Hysteria may be responsible for loss of taste, but in it, as in some cases of hemianæsthesia, there is less definiteness than a peripheral lesion gives, and the abnormality is not infrequently rather limited to want of true perception of certain taste impressions while others are retained intact, than to absolute ageusia.

It may not be amiss to refer here to perversion of taste and other functional abnormalities, and in concluding this section to describe the close relationships of smell and taste.

Hypersensitiveness for taste sensations, or hypergeusia as it is called, may be the result of irritation of gustatory nerve-fibres. Such hypergeusia has been induced by aural disease, probably as the result of stimulation of the chorda tympani nerve, and generally such taste impressions are of an unpleasant character. It is also certain that changes in the mucous membrane in which the taste bulbs are situated may be responsible for both partial loss of taste and hypergeusia. Certainly in hysteria it seems most probable that as the pharynx is often anæsthetic, the local functional changes may include local interference with taste perception.

Parageusia, or perversion of taste, is by no means uncommon in hysteria and in insanity, but it must in most if not all these cases be placed in the category of delusions, and therefore hardly demands further discussion here. Occasionally the aura of an epileptic seizure is gustatory, and may be either a powerful taste perception of some ordinary kind, indicating that the producing factor must be powerful irritation of the cells in the cerebral taste centre, less likely the gustatory nerve-fibres, or not infrequently the aura belongs to the order of totally abnormal taste impressions.

It has for many years been recognised that the senses of smell and taste are closely associated together, and many of the sensations which we are in the habit of calling taste impressions are in reality appreciated by the olfactory sense. Thus the enjoyment afforded by the aroma of a good vintage port, or the flavouring agent in any savoury dish, is almost entirely dependent upon our sense of smell; and where smell is lost, all such perception is lost with it; while on the other hand, sweetness, saltness, sourness, and bitterness may still in a case of this kind be appreciated by the sense of taste.

Atrophic rhinitis, occlusion of the nose or olfactory clefts, are common causes of anosmia, and in such cases taste must suffer. It is a frequent experience in practice to find patients who have lost their sense of smell complaining of greatly impaired enjoyment of the pleasures of the table. In one case which the author can recall to memory, as the result of atrophic rhinitis and consequent anosmia, so vitiated was the taste perception, that highly-tainted fish was eaten with relish, the patient in no way being sensible of its objectionable qualities.

It is important to remember that where anosmia has been of long standing, from whatever cause it may arise, recovery is extremely improbable.

It is unnecessary to add any further remarks with regard to prognosis or diagnosis.

The treatment of an affection of taste implies removal of the causal agent if possible. In some cases galvanism has been recommended, one electrode being placed behind the mastoid process, and the other, a flat plate of metal, being applied to the tongue, but such treatment is only likely to be beneficial in a very limited number of cases.

Spasm of the Muscles of Mastication

These spasms may be tonic or clonic, and are generally rather symptomatic than constituting in themselves a separate disease.

Tonic spasms occur in tetanus, where the lower jaw may be absolutely rigid, and the muscular contraction so excessive as to be painful. Tonic spasms may also occur in severe cases of tetany and in hysteria. Tonic spasm is not very uncommon as the result of the irritation of first dentition or caries of a tooth, and specially, it is said, the last molar may cause the condition in both the infant and the adult. Pontine tumours, hæmorrhages, or areas of softening occasionally induce very persistent and distressing spasm, apparently from irritation of the cells constituting the motor nucleus

of the fifth nerve, but other nerves or nuclei are generally involved in addition.

Clonic spasms of the masticatory muscles occur in most conditions where other muscles are similarly affected, as in convulsive seizures of all sorts, and they also occur in rigors, when the teeth are said to "chatter," in excessive demonstrations of fear, under the effect of extreme cold, and sometimes in paralysis agitans and kindred conditions. Isolated spasms are met with in chorea, where the teeth may be snapped together, sometimes biting the tongue, and that without any warning to the patient.

The prognosis depends on the nature of the spasm, and would include that of many different diseases, such as tetanus, epilepsy, etc.

The diagnosis is easy. It is only necessary to remember that tumours and inflammatory or other swellings near the articulation of the lower jaw may prevent movement, and that there are cases of rheumatoid arthritis in which the joint may participate in the changes generally better marked elsewhere, and may eventually become more or less fixed.

Treatment.—If possible we must aim at the removal of the cause of the spasm, whether tonic or clonic. Local blisters are often efficacious in hysterical cases, and bromides and other sedative remedies are worthy of trial in all cases in which hysteria can be excluded. Hot baths are generally most serviceable, and the galvanic current may be employed, with benefit in some cases, one pole on the back of the neck and the other over the affected muscles. Feeding may have to be carried on in severe tonic cases by means of a tube introduced through any gap in the teeth, or a tooth may have to be extracted or cut over, so as to permit of such feeding.

Figs. *See* Ficus.

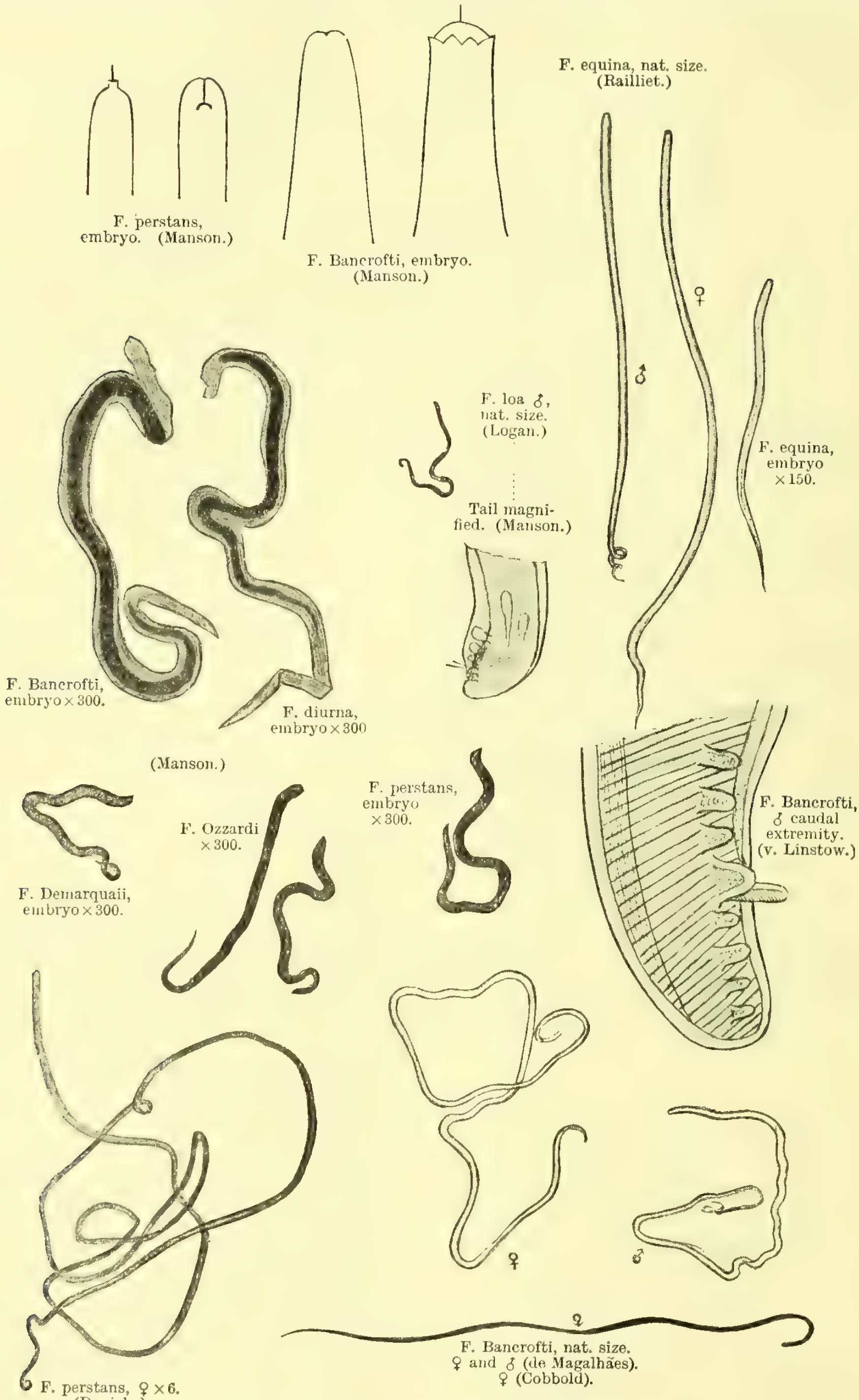
Figures. *See* Physiology, Cell (*Mitotic Division, Chromatin Figures*); Physiology, Senses, Vision (*Field of Vision, Purkinje's Images or Figures*).

Filariasis.

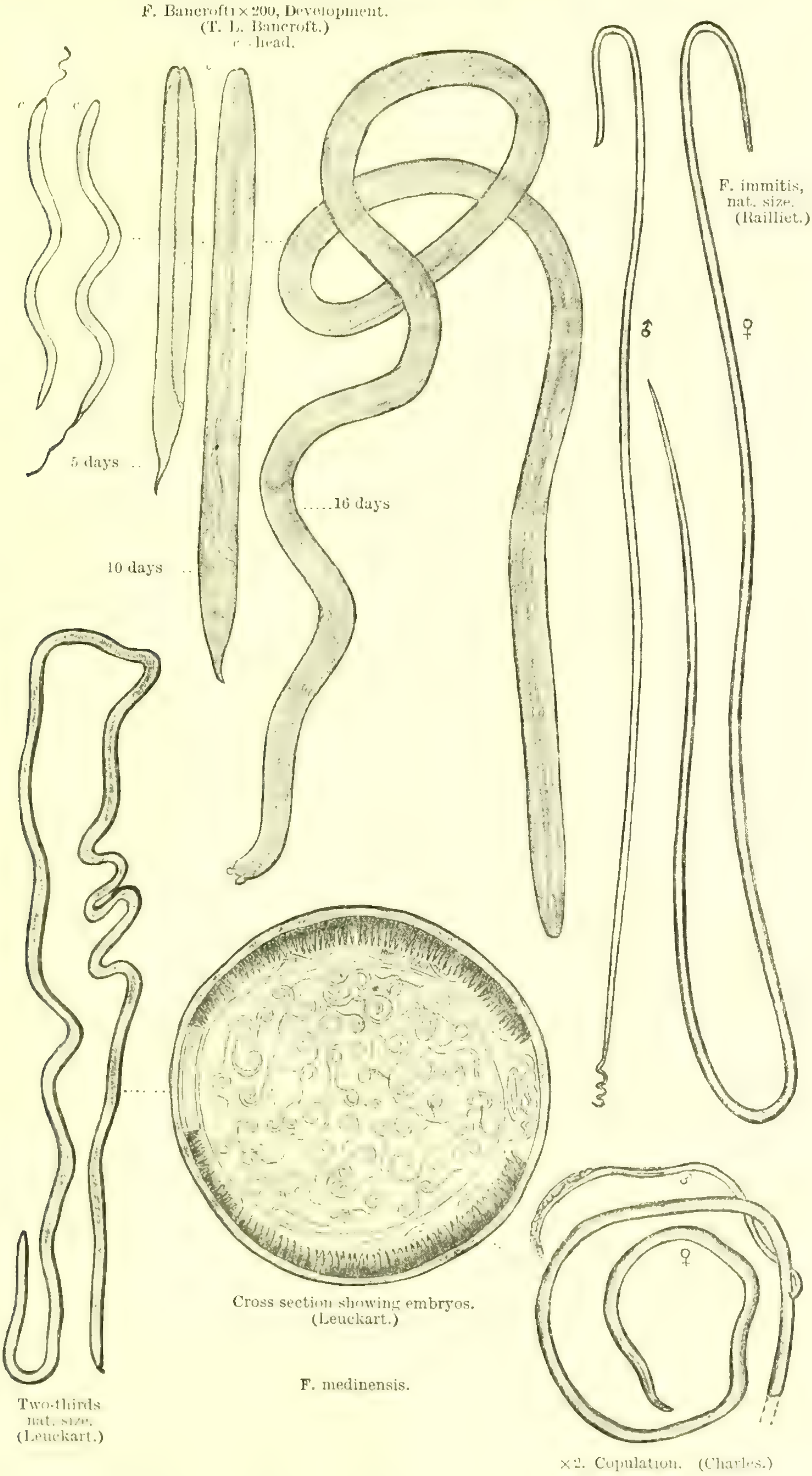
See also Ascites (*Causation, Chylous Ascites*); Hæmaturia; Parasites (*Filaridæ*); Sleeping-Sickness; Trypanosomiasis.

Introductory.—Though the term "filariasis" has been usually applied in medical works only to diseases due to *Filaria Bancrofti*, it appears advisable so to extend the term that it may include all diseases, both of man and animals, which are due to the presence and action of filariæ. Recently the term "malaria" has been similarly extended so as to include a number of diseases of animals caused by parasites more or less similar to those of human malaria, and in both cases the adoption of such a collective term seems advantageous. There is certainly no valid reason why one disease due to filariæ should be called filariasis to the exclusion of others. In the following article most attention has naturally been given to the filarial parasites affecting man, but it also seemed desirable to include some of the more important filariæ affecting animals. The latter are not included merely because of their scientific interest, but on the ground that the

establishment of facts concerning some of them may possibly serve as a guide and stimulus to further researches on the biology of the parasites affecting man. Moreover, certain filariæ (*F. medinensis, F. inermis*) are parasites common to man and certain animals.



The filariæ inhabit chiefly the blood-vessels, lymphatics, connective tissue, and serous cavities of their host. In many cases their mode of development is unknown, whilst in others it has been clearly established that they complete their life cycle through parasitism in two sets



of hosts. The host in which the filaria reaches full maturity and gives birth to its offspring is known as the definitive host, the other host being known as the intermediary or secondary host. We have examples of this double parasitism in *F. Bancrofti*, which is a parasite of man and certain species of Culex; in *F. recondita*, which is parasitic in dogs, and also in fleas and a certain species of tick; *F. rytipleurites*, affecting the rat and the cockroach;

F. uncinata, harboured both by ducks and by a small crustacean. In these examples the definitive host will be found named before the intermediary host. Reasoning from analogy, it may safely be laid down, as a general rule, for most of these parasites that may require two hosts for their complete development, the secondary host is usually to be sought for amongst insects, crustacea, or the like. There is a wide field of work open to investigators, as many of the secondary hosts have still to be discovered, and with their discovery a more accurate knowledge will be gained with regard to the mode of infection. Attractive as it may seem, the task is undoubtedly fraught with many practical difficulties. Railliet's nomenclature has been largely followed. It will be observed that certain worms have numerous synonyms; this, however, need not lead to confusion, as the name generally adopted by leading authorities is given precedence.

General Characters of the Filariæ.—The genus Filaria (O. F. Müller, 1787) comprises a large number of species which possess the following characters:—The worms are filiform, having a fairly uniform transverse measurement throughout their entire length, though some taper off gradually toward both extremities. The cephalic extremity is rounded, the mouth being often of simple construction. The males are, as a rule, markedly smaller than the females, and have a recurved or spiral tail, the latter being at times furnished with lateral membranous outgrowths, whilst they usually possess four preanal and a variable number of postanal papillæ, and spicules which generally vary both as regards appearance and size. In the female worms the vulval aperture is more or less in the vicinity of the mouth.

I. Filariæ affecting Man or Man and Animals

I. *Filaria Bancrofti*, Cobbold, 1877. Syn.: *Trichina cystica*, Salisbury, 1868; *F. sanguinis hominis*, Lewis, 1872; *F. dermathemica*, Da Silva Aranjo, 1875; *F. Wuchereri*, Da Silva Lima, 1877; *F. sang. hom. nocturna*, Manson, 1891.—The adult worm was first found by Bancroft (1876) in a lymphatic abscess of the arm and subsequently in a hydrocele, the discovery being confirmed by Lewis, Aranjo, and Manson. The last named found that it normally inhabits the lymphatic vessels, the embryos, and also at times the adult worms, gaining access to the circulation through the thoracic duct. The sexes are usually found together, six to seven worms lying intertwined (Maitland), or the parasites lie stretched out along the length of the vessels. The *Filaria sanguinis hominis*, the embryonal form of *F. Bancrofti*, was first observed by Demarquay of Paris (1863) in a man from Havana who suffered from chylocele. It was discovered in the blood and named accordingly by Lewis (1872) in India. Since then it has been established that such embryos may belong to different species, and Manson has suggested the necessity of distinguishing them by appropriate names, even though the parent forms are unknown. (See *F. diurna*, *perstans*, etc.) Wucherer (1866) found the embryos in the urine of a case of tropical chyluria, and Lewis and many other observers found them, not only in the blood, but even in the tears and secretion of the Meibomian glands in cases of chyluria, elephantiasis, or in apparently healthy subjects.

Morphology.—The adult parasite (*see* Plates accompanying this article) is filiform, actively motile, whitish and opaline in colour, with gradually tapering ends which terminate bluntly. Mouth terminal and simple. The *male* is 70-80 mm. long by 0·407 mm. broad, the delicate tail forming one or several spiral turns. According to Railliet and von Linstow (*see* Plate), it possesses four pairs of papillæ (Manson says positively that they have not been seen), and a single unpaired papilliform projection situated preanally, four (Manson says three) postanal papillæ and two unequal spicules, one measuring 170 μ. The *female* measures 155 mm. (Manson gives 94 mm.) by 0·715 mm., the vulva 2·56 mm. (Manson gives 1·2 mm.), from the cephalic extremity. The discrepancies in the measurements given by these authors are doubtless due to different modes of preparation. The vagina is very short and bifurcates into two uterine horns which run back nearly to the tail and are filled with embryos in all stages of development. The *eggs*, which are normally found only in the upper part of the uterine tubes, measure 38 μ by 14 μ. The *embryos*, which are found in the lower part of the uterus and vagina of the parent worm as also in the host's circulation, are 270-340 μ long by 7-11 μ broad, and are enclosed in a delicate membrane (remains of vitelline membrane, according to Manson), which does not impede the rapid and active movements of the worm. Owing to the retraction of the worm at either end the loose membrane may present the appearance of a lash. The tail measures about one-fifth of the entire length of the entire worm. An ill-defined granular aggregation situated axially at the juncture of the middle and posterior third of its body represents the developing intestine, which is rendered clear by staining. At a short distance behind the head a luminous V-shaped spot (water-vascular system?) is visible, the apex opening outwardly. A similar but smaller spot is observable near the tail end and the cephalic armatures, the latter consisting of a "retractile and protractile six-lipped prepuce, covering and uncovering a thick, hemispherical proboscis, which is further provided with a minute, filiform, protrusible, apical spine" (Manson).

These embryos will remain alive for days in a blood-preparation which has been sealed with vaseline and kept at room temperature. The unstained embryo is best studied when the movements begin to grow sluggish.

Examination of Blood for Embryos.—Their presence in the blood is conveniently determined by spreading a blood-film on a slide or cover-glass, drying it in the air, and staining with ordinary aniline stains. Manson recommends the making of *thick* blood films, as this facilitates the finding of embryos when few are present. It is best to examine blood which has been drawn at 8 to 9 p.m., when the parasites are more numerous. The preparation need not be fixed before staining. With this treatment the hæmoglobin is dissolved out in the dye, and only the parasites and the nuclei of the leucocytes are seen to be deeply stained. The stain (fuchsin, gentian-violet, etc.) is prepared by adding 2-5 drops of a concentrated alcoholic solution of the dye to a watch-glass filled with water. If over-stained, decolorise with dilute acetic acid. Unstained dry films may be kept indefinitely. An immersion-lens is requisite for the study of finer details.

Geographical Distribution.—The *F. Bancrofti* is very common in warm climates, especially in certain districts in tropical and sub-tropical countries. It has been observed in India (Lewis), China (Manson), Japan (Scheube, Baelz, etc.), Australia as far south as Brisbane (Bancroft), New Caledonia, it is particularly prevalent in certain of the South Pacific Islands, such as Samoa (50 per cent of people affected, Manson), and Fiji, etc., in the southern United States of America (Guitéras, Matas, Mastin, de Saussure, Slaughter, Henry), and West Indies, Guiana (Winckel, etc.), Buenos Ayres (Wernicke) and Brazil, Egypt (Sonsino), Soudan and Algeria (Cauvet), on the Zambesi and Lake Nyassa, the Zanzibar Coast (Felkin), Old Calabar, Dahomey, Madagascar, Mauritius and Réunion, etc.

Development.—The idea that the mosquito might serve as a carrier of the *Filaria nocturna* occurred almost simultaneously to Bancroft and Manson, but it was reserved for the latter (1878) to establish the important fact that the embryos of *F. Bancrofti* undergo developmental changes in mosquitoes which have ingested blood containing them. Manson allowed mosquitoes to infect themselves by sucking the blood of filarial subjects and observed the changes which took place from day to day. At first the embryos, enclosed in their delicate membrane, are almost structureless when viewed unstained. Later the tube separates from the body, giving rise to a double contour, whilst the embryo exhibits very much more marked transverse striation. The sheath is then ruptured by the worm, which butts against it with its cephalic extremity. After rupturing the sheath, the embryo bores through the stomach-wall of the insect and finds its way into the thoracic muscles, where it undergoes the following developmental changes:—It first becomes more markedly striated, then less so, the striation gives place to a granular appearance, and the movements slow down. After 36 hours movement ceases, the worm grows short and broad, the granules finer. By the end of the third day the tail appears to spring abruptly from the end of the sausage-shaped body, and large cells may be distinguished in the hitherto homogeneous protoplasm, whilst it sometimes exhibits a double contour. Indications of a mouth are seen, and, through pressure applied to the cover-glass, of an anus a little in advance of the tail. The worm now becomes elongated, the mouth acquires its papillæ, and the alimentary canal is indicated by a delicate line running from mouth to anus. Movement at this stage is feeble, being confined to the caudal appendix, which gradually disappears. As most mosquitoes died (from want of feeding, see below) on the 4-5th day after their last meal, later developmental stages were only observed in a few instances. In four cases a high degree of development was reached, the parasite being visible to the naked eye and measuring $\frac{1}{16}$ of an inch in length by $\frac{1}{825}$ in breadth, whereas the original embryos only measured $\frac{1}{90}$ of an inch. The large cells within the worm have now become smaller and are gathered around the dark line representing the alimentary canal. The characteristic valve-like termination in the intestine is developed; the mouth is open and funnel-shaped, and the tail reduced to a stump. The movements now become brisker, the body elongates, all cellular appearance vanishes, and owing to the increased transparency of the tissues, the details, except a line indicating the intestine, can no longer be made out. The tapered caudal extremity is crowned by papillæ, Manson (1883) being unable to determine if there were three or four. In this, presumably the last stage of development within the mosquito, the parasite "becomes endowed with marvellous power and activity. It rushes about the field, forcing obstacles aside, moving indifferently at either end, and appears quite at home, and in no way inconvenienced by the water in which it has just been immersed." It has been assumed that the worm bores its way out of the insect host at the time when the female insect dies after laying her eggs in the water, and that man becomes infected through drinking water containing filariæ which have developed from embryos to a condition approaching maturity in the mosquito. The filariæ then bore their way through the human intestine, and having found a suitable resting-place, attain maturity; when, fecundation having been effected, new swarms of embryos are given off within the warm-blooded host, in this way completing the cycle of development. Of the embryos that are taken

up by the mosquito many die and are digested or expelled in the fæces. Lewis (1878), who confirmed Manson's observations, followed the development in the thoracic and abdominal tissues of the insect, but he did not follow the development as far as Manson's latest stage. Sonsino subsequently (1884) observed the development in *Culex pipiens* (?) in Cairo, the parasite attaining the large form observed by Manson. He found that fleas and bugs did not serve as intermediary hosts. It having been proved that various species of mosquito can be kept alive for a considerably longer period than that mentioned above, it seemed necessary to try and follow the development, if possible, still further. Bancroft (1899) of Deception Bay, Queensland, the son of the discoverer of *F. Bancrofti*, reports that he has followed out the development of the filaria in mosquitoes. He was able to keep *Culex ciliaris* alive for two months by feeding them on bananas after they had sucked filarial blood. In his experiments, the development took place more slowly than with Manson, the last stage being only reached on the 16-17th day, and in "cold" weather on the 28-35th day. Though he does not give the temperature at which the mosquitoes were kept, it seems probable to the writer that it was owing to the low temperature that the development took place so slowly. The unimpregnated mosquito lived longest, the impregnated lived 2-3 weeks. Bancroft states that the embryos wander into the body of the insect, chiefly into the thoracic muscles, immediately after they have been sucked up in the blood. Some embryos remain behind, lose their sheath, become transversely striated, and show other signs which he refers to osmosis and the effects of digestion through the insect's secretions. We have seen that Manson described these as developmental forms. All the filariated mosquitoes (20) examined after 16-60 days contained filariæ, the number of worms present being 3-25. The insects were apparently uninjured through the filariæ. On tearing open the body of such insects under water the filariæ were liberated. They wriggled about actively but did not move—after 3-4 hours they had died. In mosquitoes that had died a natural death the filariæ were found dead after 24 hours. He never observed that the filariæ escaped from the insect's body. It seems probable from this that infection may occur (as is the case with certain other parasites) through the whole infected insect being swallowed. Bancroft quotes authority for the statement that *C. ciliaris* appears to have been introduced into Australia, and writes: "It will not go wild, but always frequents habitations, breeding in receptacles holding water in and about the house." Working with other species of mosquito, Bancroft found that the parasites did not grow in *C. notoscriptus* (Skuse), nor in *C. annulirostris* (Skuse). In the first species none left the stomach, in the second a few wandered out, but died, and were absorbed after being two days in the muscles. *C. hispidosus* (Skuse) and *C. vigilax* (Skuse) lived about seven days in confinement, and contained no filariæ after that time. *C. nigrothorax* (Macquart), *C. procax* (Skuse) and *Anopheles musivus* (Skuse) only remained alive three days, the filariæ being observed to wander into the thoracic muscles of the last-named species. In a footnote he advances the unlikely hypothesis that infection might take place when the insect containing the large developed forms is sucking blood, the warm blood entering its body, stimulating the worm, which bores its way into the œsophagus of the insect, and passes out through the proboscis into the warm-blooded host.

Henry (1896) found the embryos lived 3-4 days in blood ingested by leeches, whilst they remained alive for ten days in blood kept in a cold room. They were not killed by exposure to a temperature of 0° C. for several days. Manson states that the throwing-off of the enveloping membrane may be observed on the slide by chilling it for 6-8 hours in a refrigerator and then warming up the slide.

Periodicity.—Manson (1881) and afterwards Mackenzie, and Myers in the same year, found that the filarial embryos appeared and disappeared periodically from the blood. Manson "watched it preserve its rhythm for a month on end," that is, the filariæ appeared towards evening, increased in numbers during the night, and decreased in the morning. Mackenzie, who studied a case in London, found that if the filarial patient was kept up all night and allowed to sleep during the day the filarial periodicity above referred to was reversed, that is, the parasites appeared in the blood during the day and disappeared at night. Myers states that the embryos in the blood are very active in the earlier part of the night, and that they then grow torpid and feeble, being shrivelled and straightened out when disappearing. Manson denies the accuracy of this statement. He thought that new embryos were produced by the parent worm at regular intervals, and that they die off in the blood. The fact has, however, been established by Manson that the embryos are given off constantly into the lymph-stream. In a post-mortem examination made on a filarial patient who had committed suicide by means of prussic acid at 8 a.m., it was found that the lungs contained huge numbers of filariæ, whilst there were fewer in the carotids and heart-muscle. There were relatively very few elsewhere, and they were almost completely absent from the spleen, liver, and bone-marrow. This certainly suggests the possibility of their periodically finding a resting-place in certain parts of the body (Manson 1899). The latter author (1883) considers that "filarial periodicity is an adaptation of the habits of the filaria to

those of the mosquito, the intermediary host indispensable to the future life of the parasite."

Pathogenetic Effects.—The various symptoms and pathogenetic effects produced by this parasite have been included in most medical works under the name of *filariasis* (*see* Introduction). The period of incubation is of undetermined length. Of eighty-five persons exhibiting blood-filariæ, Manson found nine that were to all appearances healthy. The frequency with which the parasite is found varies in different places; for instance, it is found in Amoy and Bahia in 10 per cent, in Cochin in 30 per cent (Manson), in the Friendly Islands in 32·24 per cent (Thorpe 1896), and in Samoa in 50 per cent of the population. This probably depends upon the hygienic conditions (water-supply, etc.) under which the natives live, and the prevalence of a suitable mosquito to serve as a host. It has been estimated that the blood of a man may contain forty to fifty million embryos, in spite of which the host in the majority of cases suffers no noticeable effects. In other cases the parasite brings about a varicose condition of the lymphatics, producing a variety of conditions known as chyluria, hæmatochyluria, varicose groin glands, lymph-scrotum, chylocele, orchitis, endemic lymphangitis, certain forms of lymphorrhagia, and cellulitis, and probably endemic elephantiasis Arabum.

Chyluria and Hæmatochyluria occur intermittently and usually do not produce any special disturbance in the general health. The urine may be normal in quantity or increased in amount, and presents a milky, pinkish, or bloody appearance. This may vary in the same case during the course of the day. The urine may be normal in the morning, become chylous during the day, or the reverse may be observed. The affection may persist a long time. Osler cites a case of intermittent hæmatochyluria lasting eighteen years, the only inconvenience consisting in the occasional passage of hæmoglobin-tinted chyle-clots (these often cause retention) formed in the bladder, and the presence at times of uneasy sensations in the lumbar regions. According to Manson a relapse is usually preceded by dragging, aching sensations in the loins, groins, thighs, testes, and about the pelvis generally. In women the disease may make its appearance after pregnancy, whilst in man it follows any physical exertion, though often no immediate exciting cause can be determined. The patient may pass normal urine during intervals, which last either weeks, months, or years. Where the condition persists, symptoms of anæmia, including great debility and mental depression, are observable. Death may be brought about by secondary bacterial infection giving rise to deep-seated abscesses, etc. In a case described by Mackenzie, the renal and peritoneal lymph-plexuses were much enlarged, extending from the diaphragm to the pelvis, the thoracic duct being occluded above the diaphragm. *Chylous urine* generally clots quickly on standing, the coagulum as it contracts becoming pinker or redder, the fluid separating into a greasy, cream-like pellicle containing oil-globules and fatty granules, a middle layer composing the greater part of the fluid and containing fatty matter, red blood corpuscles, lymph-cells, a suspended coagulum, and, lastly, a slight dark-red sediment made up of the corpuscular elements just mentioned, clots, epithelium, salts, and usually some filarial embryos, these also being present in the clot, where, as also in the deposit, a search for them is usually quickly rewarded. Chylous urine becomes clear on the addition of ether, and on boiling gives an albuminous precipitate. The diagnosis depends upon these characters in the urine, and the presence of the parasites in urine and blood, etc.

Lymph-scrotum, according to Manson, is an affection which "is almost a sure indication of the presence, actual or past, of *F. nocturna* in the lymphatics." In this disease the scrotum is more or less enlarged, thickened, and covered with varicosities or with herpes-like vesicles 2-20 mm. across, the same containing a variable amount of serous, milky, pinkish, or red fluid, which escapes on their being punctured and may continue to drip for hours afterwards. The fluid coagulates on standing, and is seen to contain lymph-cells and blood corpuscles, etc., and generally living filariæ, which are usually also present in the blood. The disease, which is often complicated by orchitis, may last for years, inflammation and febrile attacks occurring at intervals, and being followed by lymphous discharge or abscess formation. The affection may coexist with chyluria, varicose inguinal glands, and may be followed by elephantiasis.

Lymphangitis occurs especially in elephantiasis, varicose glands, and lymph-scrotum, but is also frequently found in other forms of filarial disease. It may be limited to the abdominal organs, the inguinal glands, the testes, or spermatic cord. In the limbs it causes painful cord-like swelling about the glands and adjacent lymphatic vessels, the skin and connective tissue of the part becoming tense and inflamed. The attacks are characterised by prolonged and severe rigor, followed by high fever, and accompanied by headache, anorexia, and at times by delirium and vomiting. The attack may subside after a few days, the tension of the part being relieved by discharge of lymph through the skin. Some thickening of the tissues affected is always noticeable after such an attack, the conclusion of which is usually ushered in by copious sweating. The attacks of lymphangitis may occur with considerable regularity. For instance, in a case reported by Young (1897), the patient affirmed that the attacks had recurred every three months during the preceding six years.

Varicose Inguinal Glands (the axillary glands are rarely affected) are often associated with lymph-scrotum; they occur at times with chylous dropsy of the tunica vaginalis, with chyluria, or with all of these conditions. The glands may be affected on one side or both, giving rise to hernia-like, firm, lobulated, or fluctuating swellings, varying from the size of a walnut to that of an ostrich egg. These swellings consist of dilated lymphatics, the varicose condition extending into the pelvis and involving the thoraic duct. Tapped by means of a hypodermic needle, a fluid similar to that found in lymph-scrotum is obtained; this generally contains filariæ. These tumours, which give a dull note on percussion and little or no impulse on coughing, disappear slowly on applying pressure, and are reduced when the patient is placed in the horizontal posture. The tumours are usually painless at first, only causing discomfort through sensations of dragging and fulness, the same being aggravated by exercise and standing or walking. They are, however, at times rendered very painful through the occurrence of periadenitis.

Chylocele is a condition often associated with the above; here the tunica vaginalis contains more or less milky or red fluid, such as has been described above, whilst filariæ are often present in the blood.

All of the affections just described are brought about, according to Manson, by the occlusion of the thoracic duct, due to the presence or effects of the filariæ. The effects remain, but the worm may die. When the duct becomes occluded, a stasis of chyle and lymph takes place in the whole tributary system of the duct, and an attempt at the formation of an anastomosis with the lymphatics draining the upper portion of the body is made. To reach this anastomosis the contents of the duct must run backwards by way of the pelvic, inguinal, upper femoral, and scrotal lymphatics over the abdomen to the upper part of the body. The vessels along the route of travel become varicose, and, depending upon where they rupture, the conditions above referred to will be produced. "When we tap a lymphatic varix of the scrotum or the groin, we tap the anastomotic plexus by which the chyle, denied a route up the thoracic duct, is finding its way to the circulation."

Elephantiasis arabum is frequently encountered in tropical countries, but is practically unknown beyond 35° N. and S. latitude. Manson states that 5 per cent of the population are affected in parts of Travancore, nearly 50 per cent in Samoa and Huahine. The disease usually attacks the legs or scrotum, or both, more rarely the arms, mammæ, and female genitalia. Manson (1894) and Thorpe (1896) state that a greater liability to implication of the arms and breast is observable in the South Sea Islands. The disease begins with lymphangitis, erysipelatoid inflammation of the integuments, "prolonged rigor, followed by high fever ending within one or two days in diaphoresis," and "often in a sort of lymphous weeping from the implicated skin" (Manson). The attacks recur at intervals, and are followed by enlargement of the part, so that after some years enormous hypertrophy of the skin and cellular tissue results, giving rise to the "elephant-leg," which may measure 10-12 inches or more across, or to enormous growth of the scrotum, so that the part may weigh 20-30, or even in extreme cases as much as 200 pounds. Diseased mammæ may hang down as far as the pubes or knee, whilst the hypertrophied labia may weigh as much as 8 pounds. When the scrotum is much enlarged, it assumes a pyriform shape and rests on the ground. The integument of the penis having gradually been drawn down, the glans retaining its normal position, the latter comes to be situated at the bottom of a long tube of skin which opens about half-way down the front of the scrotum. The testes are often atrophied or deformed; they are generally, though not always, situated at the end of the greatly elongated cords, having been carried down with the growth. Hydrocele is frequent. When, as occasionally happens, the skin of the penis is affected, curious deformed enlargements resembling a ram's horn may result on the anterior surface of the scrotum. Circumscribed patches of skin may also be affected by elephantiasis. In old cases the skin is roughened and tuberose, in the case of the leg great folds of skin form about the ankle, which retains some power of motion. "The nails are rough and thick, the hairs in parts long and coarse, sensation impaired, sweating defective, skin often dark. The leg is not usually affected above the knee, the line of demarcation being either sharp or more or less diffuse. Sections of the affected skin show the derma and subcutaneous tissue to be greatly thickened, fibrous, and white, whilst the deeper layers of the superficial fascia are converted into a loose, yellowish, blubbery-looking dropsical tissue containing here and there fibrous bands, many large veins and lymphatics. The sheaths of the large vessels and nerves and the muscular aponeuroses are thickened, the underlying bones may be hypertrophied and rough, the lymphatic trunks dilated, their radicles varicose and thinned" (Manson). The lymph-glands are enlarged and fibrous. Large ulcers, abscesses, and gangrenous patches may give rise to serious complications. That filariæ bear an etiological relation to elephantiasis arabum is claimed by Manson, who advances the hypothesis that the condition is brought about by the blocking of the lymphatic vessels through filarial ova and the effects consequent thereon. He explains the fact of filariæ being rarely

found in elephantiasis as due to their being unable to pass into the circulation through the blocked lymphatics.

"Elephantoid Fever" is a term which has been applied by Fayrer to the febrile attacks which accompany elephantoid disease, the fever recurring at intervals of weeks, months, or years. Where the attacks recur frequently they may lead to a false diagnosis of malaria, from which affection it should, however, be readily distinguished by the local appearances, the prolonged fever, as well as by the absence of the malarial parasite from the blood, and the ineffectiveness of quinine.

Prophylaxis.—Water used for drinking purposes should be boiled or filtered, and it is well to keep it stored in covered reservoirs so as to exclude the possibility of its becoming infected through mosquitoes. In this connection it is advisable to cite the observation made by Thorpe (1896), who states that 40 per cent of the male population at Nomuka (Friendly Islands) are affected with filariæ, the people there drinking the water from many small lakes about which mosquitoes swarm. The chiefs, who keep their water-supply in covered tanks, very rarely or never acquire the disease.

Treatment.—Methylene-blue (recommended by Austin Flint), quinine, thymol, etc., exert no appreciable effect upon the blood-filariæ. In chyluria the patient should be kept in a recumbent position, and avoid fatty food, restricting the use of water. "A single tumblerful of milk will at once give ocular proof of the patency or otherwise of the rupture in the varix. Not until clot and albumin have entirely disappeared, and the milk test gives a negative result, should the patient be allowed to quit the recumbent position" (Manson). It is well to give a saline aperient from time to time. Manson recommends that varicose inguinal glands should be left alone or supported by a bandage. Chyluria and elephantiasis have been seen to follow operation. In lymphangitis rest, "elevation of the affected part, cooling lotions or warm fomentations, mild aperients, opium to relieve pain, and if tension is great, pricking or scarifying the swollen area under suitable aseptic conditions," are recommended. Maitland (1897) states that his experience has confirmed him in the opinion that "operative measures are called for in many cases of lymphangitis of filarial origin. Apart from the possibility of removing the parasites themselves, the operation itself relieves the patients from the annoyance caused by the recurring attacks of inflammation which so frequently incapacitate them from carrying on their duties. . . . Cases in which there have been attacks of chyluria, or in which there is other evidence to show that the obstruction to the circulation of lymph is seated high up, are not suitable for operation." Maitland reports five cases, in four of which it is stated that the filariæ persisted or reappeared in the blood. Twenty-four cases had been operated on in the Madras General Hospital during the preceding seven years. In lymph-scrotum Manson advises the same general treatment as before, but recommends excision when the condition is passing into confirmed elephantiasis. In elephantiasis of the scrotum, when the tumour is large and ungainly, removal of the scrotum, the penis and testes being retained, is frequently resorted to (*see* "Scrotum"). In cases of elephantiasis of the leg, rest, elevation, massage, and elastic bandaging are used. Mercurial treatment has been found of benefit in early cases of good constitution. In acute cases great relief is experienced after puncturing the distended limb and allowing the pent-up effusion to escape.

II. *Filaria Ozzardi*, Manson, 1897.—This filaria was found by Manson in blood sent him by Ozzard, the same having been obtained from aboriginal Indians inhabiting the back country of British Guiana. Elephantiasis, according to Ozzard, does not occur amongst these Indians, and Manson (1898) has never found embryos of *F. Bancrofti* in specimens of blood sent him by Ozzard and Daniels from the back country, whilst that parasite and elephantiasis occur along the littoral and in settled districts. Manson found filariæ in about 50 per cent of the blood specimens sent him from these parts, whilst Daniels (1898) states that he found filariæ in 134 out of 231 (58 per cent) of the natives he examined. Manson (1897), as also Ozzard and Daniels (1897), afterwards noted the occurrence of blunt and sharp-tailed filariæ, in the proportion of 5 : 1, in the blood of these natives. We have here to deal with a mixed infection, the blunt-tailed embryos belonging to *F. perstans*, the sharp-tailed to *F. Ozzardi*. The name *F. Ozzardi* was originally given to both types of embyros, as Ozzard and Daniels believed them to belong to one species. The embryos of *F. Ozzardi* (*see* Plate, p. 274) are sharp-tailed, sheathless, very small, measuring 0·173-0·240 by 0·0043-0·005 mm. (Daniels). They move about in the field as does *F. perstans*, and are also continuously present in the peripheral circulation. In two post-mortem examinations made by Daniels (1898) on natives who exhibited these embryos in their blood, he succeeded in finding groups of mature filariæ, chiefly females, in the upper part of the mesentery, the fat at its base, and about the pancreas. In one of these cases they were, moreover, found in the subpericardial fat. The male worms measured 45 by 0·06 mm., the females 70-80 by 0·12 mm. Daniels subsequently (1899) found two sets of parental forms in a native whose blood contained the two forms of embryos above referred to; the one set of parents were similar to those just described, the others are considered

by Manson to be identical with *F. perstans*, a parental form of which he observed in one of his cases of sleeping-sickness from West Africa.

III. *Filaria perstans*, Manson, 1891. Syn.: *F. sanguinis hominis*, var. *minor*, Manson, 1891. —The free embryos (*see* Plate, p. 274) were discovered before the adult forms. As their name indicates, they are found continuously in the blood, and do not exhibit any periodicity. They have no sheath, are much smaller than the embryos of *F. Bancrofti* or *F. diurna*, measuring on an average 0·23 by 0·0045 mm. The dimensions vary with their movements. Manson (1897) describes the embryos as follows:—

"The thickest part of the body is about one-third of the distance back from the head; from this point it gradually slopes off to the abruptly truncated tail. The body is homogeneous throughout, no central granular aggregation and no V-spot or other distinguishing feature being visible.[1] The head end is armed with a minute, exceedingly delicate filiform spine set on what looks like a papilla. This spine and papilla are constantly and rapidly protruded and retracted. In addition to very active wriggling movements, *F. perstans* travels about, often very rapidly, among the corpuscles, the attenuation and extension of its body facilitating its peregrinations. At no time very numerous—sometimes three or four on a slide, rarely as many as twenty or thirty—*F. perstans* can be found as readily by day as by night. It may be associated with *F. diurna* or with *F. nocturna*, or with both."

The parental forms are probably those described by Daniels (1899), who found them in a British Guiana native in whose blood both blunt and sharp-tailed (*F. Ozzardi*) embryos were present. He found a female and part of a male lying in the subperitoneal connective tissue; these were not encysted. Manson found a parental form in one of his cases of sleeping sickness. The embryos of *F. perstans* are distinguished from those of *F. Ozzardi* by having blunt tails. For further details see Manson and Daniels (1898).

Geographical Distribution.—*F. perstans* appears to be confined to the west coast of Africa and the adjoining country, in some districts as many as 60 per cent of the population being affected. It has also been found in British Guiana. Firket (1895), who examined the blood of fifty-four negroes from the Congo at the Antwerp Exposition in 1894, as also children who were being educated at Gyseghem, found filariæ in thirty (55 per cent). All the negroes seemed healthy. Some of them had not been in Africa for a year and a half, one of them had been six years in Belgium. Any pathogenetic effects which it may produce are as yet unknown.

Craw-craw, a disease described by O'Neil in 1875, is characterised by a papular and pustular eruption resembling scabies; it occurs on the west coast of Africa. The papules occur singly, in rings, or over the whole body, become converted in two days into vesicles and then into pustules. Actively moving filariæ were found by O'Neil in the papules, the worms measuring 0·01 by 0·002 inch, and bearing two black marks at the head end. According to O'Neil the affection is said to be contagious, appearing after an incubation period of three days. He states that it disappears in cold climates, but returns when the negro returns to a hot climate. (This would indicate the possibility of the worm wandering inward, as suggested by Railliet for *F. hæmorrhagica*). Sulphur inunctions have been found useless in the treatment of this malady. Manson (1898) considers that O'Neil may have had to do with *F. perstans*, which is so frequent in the blood in the regions referred to. A similar disease has, however, been described by Nielly (1882), who observed it in a boy who had never been outside France. Nielly found embryos in the blood, whilst the parasites in the skin were apparently more highly developed, possessing an alimentary canal and rudimentary sexual organs.

Sleeping Sickness.—*F. perstans* has been found in three cases of this disease. From the fact of the frequent occurrence of this parasite in negroes from the Congo and certain other places in West Africa, the etiological significance of the worm seems doubtful. (*See* Trypanosomiasis.)

IV. *Filaria Demarquaii*, Manson, 1897.—Found in the blood of apparently healthy natives of St. Vincent and St. Lucia, W.I., only the embryos (*see* Plate) being known. They resemble those of *F. Bancrofti*, except that they are smaller, have no sheath, and are constantly present in the blood. Named after the discoverer of the *F. sanguinis hominis*.

V. *Filaria diurna*, Manson, 1891. Syn.: *F. sanguinis hominis*, var. *major*, Manson, 1891. —Only the free embryos of this parasite (*see* Plate) are known, the same having been observed by Manson in the blood of negroes from the west coast of Africa, Old Calabar, and Dahomey. They differ from those of *F. Bancrofti* only in that they appear in the blood during the day and disappear at night. It has therefore been assumed that they undergo development in another intermediary host as yet unknown. *F. diurna* appear in the peripheral circulation at about 8 A.M., increase till mid-day, decrease toward evening, and disappear at 9 P.M. This periodicity has been observed by Manson to be maintained for weeks. He gives reasons for

[1] Dr. Manson has informed me recently that a minute V-spot is visible by careful examination, especially of stained specimens.

supposing that *F. loa* (see below) may constitute the parental form. No pathogenetic effects due to these blood-filariæ have yet been observed.

VI. *Filaria medinensis*, Velsch. Syn.: *Vena medinensis*, Velsch, 1674; *Dracunculus Persarum*, Kämmfer, 1694; *F. medinensis*, Gmelin, 1789 (Guinea-worm, Ver de Médine, Dragonneau).—This worm has been known from ancient times in Arabia, Persia, and other countries, the disease produced being termed "Dracontiasis" by Galen. It occurs chiefly as a parasite in man, being situated usually in the subcutaneous and intermuscular connective tissue. It has also been found in animals (Railliet). According to Avenzoar and Marchais it often affects cattle. In India it has been found in the horse (Clarkson, Fleming, Burke), also in the dog in Buenos Ayres and Curaçao (Doerssel), India (Smyttan, Forbes, Griffith), Egypt (Clot Bey, Piot, Innes), in the cheetah (*Filaria æthiopica*, Valenciennes, 1856), *Canis lupaster*, jackal, and wild-cat (Railliet). In some places at certain seasons nearly 50 per cent of the population may be affected. Usually but a single worm inhabits the host, but when the worm is prevalent two to four may be found, or exceptionally as many as thirty to fifty.

MORPHOLOGY.—The adult female[1] (*see* Plate, p. 275) may attain to a great length in the body of the host, measuring 1-6 feet, though on an average it has a length of 3 feet. Railliet states that it may even attain a length of 4 metres. The worm is of a white or yellowish colour, of uniform diameter (0·5-1·7 mm.) throughout. It tapers slightly towards the head, which is truncated. The mouth is terminal, and is surrounded by two large and six small papillæ. The body exhibits faint transverse striation. The body of the worm is firm and very extensile, having, according to Manson, a breaking strain of about 11 oz. The cuticula is thick, being formed of about six layers, the muscular system being similar to that of Ascaris. The tail is terminated by a sharply bent hook about 1 mm. or more in length. The alimentary canal consists of a fine tube running from the triangular mouth to near the tail, but it does not open externally in the gravid worm, though an anal orifice exists in the young parasite. In the mature worm the vulval opening and vagina are likewise obliterated by the enormously developed uterus, which forms a continuous tube, filled with millions of free embryos, and running from head to tail. The embryos, which *in utero* usually lie curled up on themselves, measure 0·50-0·75 μ by 15-25 μ. They are slightly flattened dorso-ventrally, transversely striated, and provided with a finely tapering tail which measures about two-fifths of their whole length. They have a rudimentary alimentary canal, and carry two small lateral sack-like structures at the base of the tail. They swim actively, and may live for days in muddy water or damp soil, whilst they are capable of withstanding desiccation for from six to twenty-four hours. To quote Manson (1897):—

"When the parent Dracunculus approaches maturity, she begins to move slowly through the tissues, head first, and in 90 per cent of the cases in a downward direction, until her head arrives at foot, ankle, or leg. The head then drills a small hole in the derma, sparing the epidermis. Over this hole a bulla forms, probably induced by the irritating properties of some secretion or discharge of the worm. In the course of a few days the bulla ruptures, disclosing a small superficial ulcer with the aforementioned minute hole in its centre. Sometimes, on rupture of the bulla, though this is by no means usual, the head of the worm is seen protruding from this little hole. If now, whether the head protrude or not, a little cold water be poured on the limb in the vicinity of the dracuncular ulcer, a minute quantity of a whitish fluid is presently seen to well up from the central hole; or a small tube, at first pellucid, then white, is seen to be slowly extruded from this hole to the extent of about half an inch or even more. Suddenly this little tube ruptures, its contents being spilt over the ulcer. The whitish fluid alluded to is part of the uterine contents, and the little tube is part of the uterus itself, which the worm, stimulated by the water poured on the limb, forces as a prolapsus through her mouth. That this is what takes place is proved by a microscopic examination of the fluid referred to, which consists of a mass of seething, wriggling, Dracunculus embryos. This discharge, with or without visible prolapsus of the uterus, is repeated at short intervals. In the course of about a fortnight the worm has extruded and emptied her entire uterus. She is now ready to quit her host, and often she will do so spontaneously. Generally she can be readily removed by gentle and intermittent traction renewed at intervals during a day or two.

"The reason why instinct leads the Dracunculus to descend to the foot or ankle before beginning to empty her uterus is obvious. These are just the parts of the body which in a warm climate, and in natural conditions of life, are most likely to be brought in contact with water. The reason why the extrusion of embryos is provoked by contact of the limb with water is equally obvious, for the next step in the development of the embryo is made in this element. It is true that the Dracunculus some-

[1] Charles (1892) twice observed what appears to be copulation (*see* Plate) in worms removed from the subperitoneal connective tissue. Nobody else has observed the male. Neumann (1895), from his observations on *F. dahomensis* (parasitic on *Python natalensis*), believes that Charles must have actually seen the male *F. medinensis*, and that the latter is also parasitic like the male *F. dahomensis*.

times reaches the surface at other points of the body, but such an event is comparatively rare and manifestly abnormal."

Development.—According to Fedschenko the embryos undergo development in a fresh-water Cyclops. If placed in water containing the Cyclops, they attack the latter and penetrate into their body cavity, through the interarticular membrane between the abdominal plates. When swallowed by the Cyclops the filariæ are digested, whereas in the body cavity they undergo a metamorphosis which consists at first in the further development of the intestine. On the eleventh day they moult and exhibit a very changed appearance, being shorter (0·5 mm.), non-striated, etc., and in four weeks have attained a length of 1 mm. Infection experiments carried out by Fedschenko, wherein he fed dogs and cats with infected Cyclops, gave negative results. Still the opinion prevails that this is the mode of infection. Subsequently Manson was able to confirm these observations in London, where he noted a slow development of the embryos (obtained from Lascars at the Seamen's Hospital) in Cyclops obtained from ponds in London. It will be remembered that Chapotin (1810) claimed, as many have done since, that the embryos are capable of entering the body through the skin. The fact that the embryos are capable of boring their way into Cyclops is certainly suggestive, and does not render this older view as yet untenable. Plehn (1898) reports that he fed two monkeys on bananas which he had infected with embryos, and that one of these animals subsequently developed a painful tumour on the thigh, and died after eight and a half months. The tumour was found to contain a worm in all respects identical with *F. medinensis*, though it only measured 40 cm. in length. The experiment is certainly most suggestive, and deserves repetition.

Geographical Distribution. — The worm occurs in parts of India, Persia, Bokhara, Turkestan, and Arabia, along the coast of the Red Sea, in tropical Africa (Guinea, Senegambia, Darfoor, Senaar, Abyssinia, Nubia, Egypt), in Brazil, Guiana, and Curaçao, whither it was carried by negroes from Africa. Railliet draws attention to the curious fact that the worm is often found amongst carnivora in Lower Egypt, whilst it does not seem to have as yet become acclimatised to the indigenous population.

Pathogenetic Effects.—Age, sex, and race exert no influence on the susceptibility to infection from this worm, except in so far as the chance of exposure varies according to habits of life. According to Duke the appearance of the worm is usually heralded by the appearance of a transient urticaria and vomiting, whilst a sensation as of a cord beneath the skin may also be noticeable. The presence of the worm is not recognised until it appears beneath the skin, which usually takes place on the lower limbs, though the worm may also show itself on the perinæum, genitalia, trunk, arms and hands, tongue and eyelids. In addition to the effects above noted, a tumour followed by single or multiple abscesses may develop at the spot occupied by the worm, the abscesses opening spontaneously. Complications of a grave nature, septic infection, gangrene, etc., may result, making amputation of a limb necessary, or even leading to a fatal termination. If bungling attempts at extraction of the worm are made, which lead to the rupture of the worm and liberation of the contained embryos, severe constitutional symptoms, with the local development of abscess, sloughing, etc., may follow. In some cases the worm dies without gaining access to the surface of the skin. It may then become cretified, and in that case is best left alone (Railliet, Manson).

Treatment.—Though the expulsion of the worm may take place spontaneously, it is in most cases necessary to extract it. This requires caution, and is best accomplished by the application of water-dressings to the part when the worm first appears. Manson recommends douching the parts several times a day with cold water, as this encourages the expulsion of the embryos, and hastens the spontaneous exit of the parent worm. An old method practised in Persia and Africa, with the object of hastening the exit of the worm, consists in placing the projecting extremity of the filaria in the fork of a small split stick and gradually winding the worm out, great care being taken not to do this too rapidly, the operation taking several days. Emily has recently recommended a mode of treatment which consists in injecting a 1 : 1000 solution of bichloride of mercury along the track of the worm, with the result that the filaria is killed and subsequently absorbed. Where the worm has been mechanically removed, an occlusive dressing may be applied, and either iodoform or oxide of zinc dusted over the fistulous opening. Plehn (1898) saw openings thus treated heal up in two to three days.

Prophylaxis.—Water used for drinking purposes should be filtered or boiled. The possibility of infection occurring whilst bathing is to be considered.

VII. *Filaria loa*, Guyot, 1778. Syn: *Dracunculus oculi*, Dies., 1860; *D. loa*, Cobbold, 1864.—This parasite is found in the subcutaneous areolar tissue, but may wander about the body, causing itching and local irritation when it approaches the skin. It is also found in the eyelids, or beneath the conjunctiva, causing severe pain, especially at night, together with congestion and lachrymation, inflammation often resulting from rubbing the parts. The symptoms may only last two to three days, when the filaria wanders away again, after which it may not cause any trouble again for weeks.

The attacks may recur during the course of years, until for some unknown reason the parasite disappears. Blot has observed that the filaria may wander from one eye to another, and it has even been observed in the fingers. Plehn (1898) considers that certain sharply defined inflammatory cutaneous affections observed in Cameroon and German East Africa may be caused by this worm. The affected areas alter their position, and are accompanied by a peculiar creepy feeling, the absolute diagnosis being impossible unless the worm shows itself. The spots affected are 1½ or more inches across, and painful on pressure. They advance 2-3 cm. a day at the margin, the affection passing away spontaneously or after three to four days' vigorous use of mercurial ointment.

Morphology.—The anatomy and life-history of the worm are imperfectly known. It usually measures 30-40 mm., and is about as thick as a fine fiddle-string. The cephalic extremity is blunt, the tail pointed, the mouth prominent and simple. The ripe eggs measure 35 by 25 μ, and contain embryos 210 μ long. Manson thinks *F. loa* may be the parental form of *F. diurna*. He describes and figures a male (*see* Plate) which measured 25 by 0·3 mm., had a simple mouth, and an incurvated tail bearing two rather short unequal spicules, and at least five pairs of papillæ, four of these being very large. The chitinous integument is sprinkled over with numerous hemispherical bosses, as in the female. (*See also* Ludwig and Saemisch, 1895.)

Geographical Distribution.—*F. loa* is indigenous to the west coast of Africa (Guinea, Angola, Gaboon, Congo), its specific name "Loa" being that given to the worm by the Congo natives. It was transported by negroes to the West Indies and Guiana, in whose persons it reappeared during five to ten years after they had landed. It does not seem to have gained a foothold there.

Treatment.—Excision. Manson states that some tribes of negroes drive the worm from the eye by placing some grains of salt in the conjunctival sac, whilst others remove it by means of a sharp thorn. Roth (1896) reports that the natives on the Niger apply a raw onion to the eye to drive away the worm. Plehn (1898), who observed the worm in Cameroon, used cocaine to lessen the pain in the eye, and, after excision, applied compresses, followed by lead-lotion. The local symptoms subsided in from three to four days.

VIII. *Filaria lentis*, Dies., 1851. Syn.: *F. oculi humani*, von Nordmann, 1832.—Under this name are included nematodes, probably belonging to different species, and varying in size from 1·72 to 12·6 mm. Railliet considers that they may represent worms which have gained access to the wrong host, or such as have been arrested in their development.

IX. *Filaria inermis*, Grassi, 1887. Syn.: (?) *F. palpebralis*, Pace, 1867, nec Wilson, 1844; *F. peritonæi hominis*, Babes, 1880; *F. conjunctivæ*, Addario, 1885.—This worm, of which only the female is known, has been found in man, the horse and donkey. In the horse it has doubtless often been taken for *F. equina*, according to Grassi. It was first observed by Dubini in the human eye, and has been found only three times. Babes found it encysted in the gastro-splenic omentum, and Addario encysted in the ocular conjunctiva. *F. lentis* (Dies.) may only be a young form of this worm. The latter measures 160 mm. by 0·475 mm. It is of a whitish or brownish colour, slightly flattened, threadlike, and tapers somewhat toward both extremities, especially at the posterior end, the tail being incurvated at its extremity. The integument is traversed by longitudinal and transverse striæ, which are, however, absent at the cephalic extremity. The mouth is very small, unarmed, and terminal. The anal aperture is 300 μ distant from the tip of the tail, the vulva 50-104 μ from the mouth. The eggs are hatched in the uterus; the free embryos measure 350 μ by 5·5 μ, and are slightly tapering in front, whilst posteriorly they end in a very fine tail.

X. *Filaria volvulus*, (?) Leuckart.—Found in two tumours, the size of a pigeon's egg, removed from the scalp and chest of a Gold Coast negro. The tumours contained tangled masses of male and female filariæ, measuring 60-70 and 30-35 cm. respectively, which are bathed in fluid containing sheathless embryos resembling *F. nocturna* and *diurna*. The tumours seemed to communicate with the outside, not with the circulatory system.

XI. *Filaria Magalhaesi*, Manson, 1897.—Found by Magalhaes of Rio de Janeiro, who described two adult worms, but not the embryos. The worms were included in a clot of blood said to have come from the left ventricle of a child's heart. They were white, opalescent, and traversed by delicate striæ. The head was club-shaped, the mouth terminal, round, simple, without papillæ. The male measures 83 by 0·4 mm. (greatest breadth), has two spicules projecting from the cloaca, distant 0·11 mm. from the tip of the rounded tail, which bears four pre- and three post-anal papillæ. The female measures 155 by 0·71 mm. (greatest breadth), has its vulva situated at a point distant 2·56 mm. from the mouth, whilst the anal aperture is found 0·13 mm. from the tip of the rounded tail.

XII. *Filaria labialis*, Pane, 1864.—Only the female is known; it was found but once, at Naples, in a small pustule on the inner side of the lip. The worm measured 30 mm., and had tapered ends, but slightly swollen quite at the posterior extremity. Mouth surrounded by four papillæ, anus distant 0·5 mm. from caudal end. Vulval aperture 2-5 mm. higher up. Uterus double.

XIII. *Filaria hominis oris*, Leidy, 1850.—Found in the mouth of a child at Philadelphia. Thought by Leidy and Leuckart to be possibly a young immature *F. medinensis.* Worm 14 cm. long by 0·16 mm. wide. Mouth simple, round. Tail blunt, and furnished with a short hook measuring 12 μ across at its base and 50 μ in length.

XIV. *Filaria lymphatica*, Treutler. Syn.: *Hamularia lymphatica*, Treutler, 1793; *F. hominis bronchialis*, Rud., 1819; *F. lymphatica*, Moquin-Tandon, 1860.—Found by Treutler in hypertrophied lymphatic glands, and in 1879 by Zahn. Diesing and Weinland consider it identical with *Strongylus longivaginatus* (*paradoxus*). Railliet thinks it more probably a male *F. inermis.* A filiform worm, slightly flattened laterally, measuring 26 mm., brown in colour, with white spots, and almost transparent posteriorly.

XV. *Filaria restiformis*, Leidy, 1880.—Probably a pseudo-parasite (Railliet); expelled in the urine of a labourer in America.

II. Filariæ affecting Animals

XVI. *Filaria equina*, Abildgaard. Syn.: *Gordius equinus*, Abildg., 1789; *F. equi*, Gmelin, 1789; *F. papillosa*, Rud., 1802; *F. equina*, Blanchard, 1849.—*F. equina* is found in the peritoneal cavity of the horse, donkey, and mule, and may pass into the tunica vaginalis, producing hydrocele, orchitis, and peri-orchitis; it is situated between the testicle and epididymis (Ercolani, Schmidt and Pottinger, Owen and Schmidt). It was once found by Gourdon in a Fallopian tube. Less frequently it is found in the pleural cavity (Mengers). Abildgaard found it between the dura and pia mater, Rudolphi in the intestine, and it was found in the liver by Sonsino in Egypt and Colin in Alfort. The filariæ which occur (usually in the aqueous humor) in the eye of the horse are generally referred to this species. Neumann (1897) concludes from the examination of thirteen eye-filariæ collected from the horse that these only represented young forms of *F. equina.*

Morphology, etc.—The worm is whitish, filiform, with tapering ends, more especially toward the tail. Integument shows fine transverse striæ. Mouth small, round, and furnished with a chitinous ring, the margin of which supports laterally two crescentic lips, and forms a simple or indented papilla at a point corresponding to the dorsal and ventral median lines, whilst posterior to these are four submedian papilliform chitinous spinules. The *male* (*see* Plate, p. 274) is 6-8 cm. long, has a spiral tail, and exhibits four pre- and four post-anal papillæ. The *female* measures 9-12 cm., has a slightly spiral tail terminating in a rounded button, in front of which are two lateral conical protuberances. The worm is viviparous, the *embryos* measuring about 280 μ by 7 μ. The development is unknown, though it probably takes place as in *F. Bancrofti*, in an insect host. According to Deupser (1892) the embryos die in water, serum, or humor aqueus in three to six hours, though they remain alive for thirty-six hours in horse's blood. Railliet finds them very sensitive to desiccation. The co-existence of embryos in the blood and adult *F. equina* in the horse's peritoneum was observed by Wedl (1848) and Sonsino (1876). Deupser (1894) failed to find the embryos in the urine or digestive tract. He introduced gravid *F. equina*, with proper antiseptic precautions, into the peritoneum of rabbits, and saw embryos appear in the blood after thirty-four hours, and persist there up to thirty-one days. In the horse's peritoneum, the female worms are more numerous than the males (as 23 : 1 Deupser), in the eye as 5 : 8 (Neumann), which would indicate that the eye is not a suitable habitat.

Geographical Distribution.—Frequent in East India, rare in America and Europe.

Pathogenetic Effects.—Usually the worms produce no symptoms through their presence in the serous cavities. Steele, however, attributed the cachexia observed by him in a horse to the presence of an enormous number of *F. equina* in its peritoneum. When present in the eye, ophthalmia results. In such cases one to two parasites, rarely more, are present, and swim about in the aqueous humor. If left untreated permanent opacity of the cornea usually results. Baruchello describes another affection which is probably due to this parasite, *i.e.* cutaneous tumours from the size of a millet-seed to that of a pigeon's egg, occurring in spring and summer in horses belonging to a cavalry regiment. The tumours softened, and nematodes were found in the pus. In three horses that died of the disease *F. equina* was found in the peritoneum. Treatment consists in the extraction of the parasite from the eye, the only situation in which it may become visible.

XVII. *Filaria hœmorrhagica*, Railliet, 1885. Syn.: *F. multipapillosa*, Condamine and Drouilly, 1878, nec *F. multipapilla*, Mollin, 1857.—Drouilly (1877) discovered this parasite in the horse. It was found in the subcutaneous intermuscular and interfascicular connective tissue and spinal cord of a donkey by Railliet and Moussu (1892).

Morphology.—The worm is described by Railliet as white, cylindrical, slightly tapering at the ends, particularly at the posterior end. The anterior conical extremity is retractile. The integument is traversed by numerous transverse ridges, which go over into many papilliform projections at the conical portion near the simple circular mouth. The *male* measures 28 mm. by 0·260-0·280 mm., and has a rounded tail, and two spicules measuring 680-750 and

130-140 μ respectively. The *female* measures 40-56-70 mm. by 0·420-0·440 mm. The tail is blunt and rounded; the vulva is near the mouth. The *eggs* measure 52-58 by 24-33 μ, and contain an embryo. The development is unknown. Fertilisation doubtless takes place in the connective tissue of the host. The female lives two to three days in serum, under which conditions some eggs are seen to hatch, the free *embryos* measuring 220-230 by 9-11 μ. The embryos are killed by drying, and live but a short time in water. Inoculation of a horse with such embryos gave no result. Railliet and Moussu think that the female expels the embryos at the time when she produces the hæmorrhages, the embryos being taken up by some insect or crustacean. Railliet believes that the worms penetrate into the deeper tissues during cold weather.

Geographical Distribution. — *F. hæmorrhagica* occurs in Asia and Eastern and Southern Europe. It would seem as if the affection caused by the worm has been known a long time in China. Sibald says it is common in Tartary. Spinola considers it a disease of the Steppes, and Leblanc observed it in Russian horses. In France it has only been known since horses have been imported from Bohemia, and it has been noted that the disease disappears in these animals after a sojourn of three or four years in France. Bernard and Liautard claim that they have seen the disease in Spanish horses in Algeria.

Pathogenetic Effects.—The disease is only noticeable in spring and summer, and is marked by the appearance of hemispherical protuberances about the size of a nut beneath the skin. These protuberances burst within one or two hours after they are formed, and allow blood to escape, after which they usually subside and appear again in from twenty-four to forty-eight hours at another place a few centimetres distant. Suppuration is rare. The donkey in which Railliet and Moussu found these parasites had come from Bohemia, and had to be killed in consequence of its developing paralysis of the lower portions of the body. At the post-mortem examination the tracks made by the worm could be seen in the donkey's cord.

XVIII. *Filaria immitis*, Leidy, 1856. Syn.: *F. canis cordis*, Leidy, 1850; *F. papillosa, hœmatica canis domestici*, Gruby and Delafond, 1852.—This parasite is found in the dog, in which host it was first seen by Panthot in 1679, and afterwards by Peyronnie (1778), and many others since. It has also been found in the fox (Sauzade), the wolf by Janson (1892) in Japan, and in man (?) by Bowlby. The adult worm is found chiefly in the right heart of the dog. Thousands of embryos are given off by the female and circulate in the blood. The embryos exhibit to a certain extent the periodicity observed by Manson in the case of *F. Bancrofti* embryos, the parasites being most numerous in the blood at night. Manson found that the embryos accumulate in the large vessels of the abdomen and thorax during the daytime. Both Grassi and Sonsino observed nematodes in the intestine and body-cavity of dog-fleas, which they thought were embryos derived either from *Filaria immitis* or *Spiroptera sanguinolenta.* It was subsequently determined that the latter parasite does not give off hæmatozoal larvæ, whilst Grassi (1888) conclusively proved that neither *Pulex serraticeps, Hœmatopinus*, nor ticks (*Rhipicephalus siculus*, Koch) served as hosts for the embryos of *F. immitis.* Sonsino was led astray, Grassi claimed, by the coincidence that *F. recondita* (Grassi) was present in the dogs he examined, and he took the embryos of the latter for those of *F. immitis.* Consequently the evolution of *F. immitis* remains to be determined. Grassi thinks the intermediary host may be a crustacean or mollusc.

Morphology.—*F. immitis* is a filiform, whitish worm, tapering at the extremities, especially at the tail, and rounded anteriorly. The mouth is terminal, small, and simple, being surrounded by six small, indistinct papillæ. Anus near end of tail. *Male* worm (*see* Plate) 12-18 cm. long by 0·7-0·9 mm. wide, with spirally wound tail bearing two small lateral ridges supported by papillæ, four of which appear larger, whilst three of them are pre- and one post-anal. According to Schneider there are eleven papillæ, of which six are post-anal. Two spicules of unequal length. The *female* (*see* Plate) is 25-30 cm. long by 1-1·3 mm. wide. The tail is short and blunt, the vulva is near the origin of the intestine, and is situated at a distance of about 7 mm. from the mouth. The *eggs* hatch in the uterus, liberating embryos about 285-295 μ long by 5 μ wide, their anterior extremity being somewhat tapered, whilst posteriorly the body tapers off into a long and delicate tail. (Railliet figures the male, female, embryos, and the posterior extremity of the male.)

Geographical Distribution.—The parasite is very common in China, where almost all the dogs are affected (Somerville), whilst over 50 per cent of the dogs in Japan harbour the worm. It is also frequent in Italy, especially in marshy districts about Pisa and Milan. It is also found in England, France, Denmark, Germany, Australia, Fiji, the United States, and Brazil.

Pathogenetic Effects. — Dogs are most affected in the period from April to August. House dogs rarely exhibit the worm, it being most common in hunting-dogs, or such as roam about the country. Though usually situated in the right heart, the worm may be found in various parts of the venous and arterial system, in the thoracic cavity, bronchi, œsophagus,

liver, in vomited matter, etc. Even where the females lie encysted outside the vessels the embryos make their way into the circulation. The parasites inhabiting a single host may number from one to fifty, the males usually being present as one to two or three females, whilst at other times only males or only females can be found. The mass of tangled worms may at times obstruct the circulation within the cavities of the heart.[1] The lesions produced by the worms vary very much according to the part occupied. When situated in the heart, they usually produce hypertrophy, and more or less endocarditis, whilst in the vessels they may produce endarteritis, thrombi, etc. According to Galeb and Pourquier the embryos may pass into the fœtal circulation, but it appears as if they did not develop there. The symptoms naturally vary greatly; weakness, anæmia, cough, icterus ascites, and lameness may be observed, and a fatal result is frequent. No effective treatment is known.

XIX. *Filaria recondita*,[2] Grassi, 1890.—The embryos of this parasite are known as the "Hæmatozoa of Lewis" (Grassi). Only a female of this parasite is known, having been found by Grassi, after much difficulty, lying (not encysted) in the adipose tissue near the hilum of a dog's kidney. The female was not quite mature. The embryos had been observed by Gruby and Delafond, who estimated that 11,000 to 224,000 might be present in a single dog. The embryos live about ten days in blood kept at 15° C. If injected into the circulation of dogs they disappear after from eight to forty days, or may persist in the blood for years. Injected into rabbits, they disappeared in about a month. In Paris 4-5 per cent of the dogs were found to harbour the parasite, whilst in China, Calcutta, and Pisa about 30 per cent of all dogs are affected (Gruby and Delafond, Manson, Lewis, Sonsino). The parasite produces usually no pathogenetic effects, but the two authors named above noted epileptiform seizures in three dogs, two of which died. No effectual treatment is known.

The female worm is 3 cm. long by 0·178 mm. wide. It is transparent, tapers (more posteriorly) toward the extremities, which are blunt, whilst anteriorly four small papillæ are seen close to the mouth. The posterior extremity exhibits one terminal and two lateral papillæ, as also small lateral projections. The mouth is succeeded by a very short cylindrical pharynx. The anus is 228 μ distant from the tip of the tail. The uterus is double (it contained neither eggs nor embryos), whilst the vulva is about 840 μ behind the mouth. Integument smooth. In 1888 Grassi observed that a great resemblance existed between the embryos of this worm, as found in fleas, and those described by Manson as occurring in the mosquito (*Culex pipiens*), which the latter regarded as stages in the development of *Filaria Bancrofti*. Grassi saw thirty to fifty embryos in a single flea, and found them in various stages of development, both in the intestine and body-cavity of the insect. The parasites also exhibited the sausage shape and the form with three caudal papillæ, such as Manson observed in *F. Bancrofti* embryos developing in mosquitoes. As Grassi (1890) was able to determine, the "Hæmatozoon of Lewis" undergoes a metamorphosis in the dog-flea (*Pulex serraticeps*), in the cat-flea (which some consider to be but a variety of the former), in *Pulex irritans*, which is often found on dogs, as also in a tick (*Rhipicephalus siculus*, Koch). The embryos of this filaria, which were first accurately described by Lewis (1875), were found by Grassi to perforate the intestinal wall of the flea, which had ingested blood containing the parasites. The latter then made their way into the fatty tissue, where they are almost always to be found lying singly in the fat-cells. These cells increase in size with the growth of the parasites, which lie curled up within the cell, its nucleus remaining uninjured. Grassi believes the worms may also undergo development outside of cells, and he observed them lying within the eggs of gravid fleas, as also in the cysticercoid of *Dipylidium caninum*. The worm-embryos undergo four stages of development, within the flea, in which insect Grassi and Calandruccio especially studied their development until almost completed. Infection experiments with fleas gave no positive result, which may have been due to the parasites not being fully developed. Grassi dwells on the resemblance between these observations and those made by Manson on *F. Bancrofti*, and figures the developmental changes above described.

XX. *Filaria irritans*, Rivolta. Syn.: *Dermofilaria irritans*, Riv., 1884; *F. irritans*, Railliet, 1885.—Found by Rivolta (1868) in the "summer sores" or "granular dermatitis" of a horse, and subsequently by Laulanie (1884). The worm also affects donkeys. The disease, which is observed in summer and in hot countries, was described by Bouley in 1850. It is characterised by the presence ("au milieu de bourgeons charnus") in the centre of fleshy protuberances of granulations the size of a millet-seed or pea, which are composed of a cheesy or calcareous substance. These granulations contain the worm, its remains, or the remains of its former habitat. The sores spread rapidly, persist a long time, and are the seat of continued pruritus,

[1] A. E. Shipley (*Proc. Cambridge Phil. Soc.* viii. pt. iii. 1894) reports an interesting case in this connection, and gives a figure of the heart of a dog from Fiji.

[2] Sonsino (personal communication, Dec. 1899) considers it doubtful that this is a "good species," the determination having been made upon a single female specimen.

being often followed by grave complications (Railliet).

Filaria palpebralis, Er. Wilson, 1844.—Found in the excretory ducts of the lachrymal glands, and under the eyelids in the horse, where they produce the same effects as *F. lachrymalis.* Railliet placed a female containing embryos in a horse's eye, but the embryos did not develop.

Filaria Evansi, Lewis, 1882.—Found by Evans at Madras in an autopsy on a dromedary, the blood of which contained embryos similar to those of *F. Bancrofti.* The adult worms were in the lung and mesentery, the pulmonary arteries being obstructed by masses of tangled worms. Both the males and females are known. Goubeaux (1853) found filariæ in a dromedary which died at Alfort in France, the worms being present in the lymphatic glands, one lachrymal gland, lung, blood, etc. Piot (1886) found filariæ in the testicle of a camel in Egypt.

Filaria labiato-papillosa, Alessandrini, 1838. Syn.: *F. cervina*, Duj., 1845; *F. terebra*, Dies., 1851.—Found in the peritoneal cavity of cattle and various deer (*Cervus elaphus, C. columbianus, C. virginianus*). Railliet considers the nematodes observed in the eyes of cattle by Grisoni, Deguillème, Santin, etc., to be fitly placed provisionally under this species. The worm greatly resembles *F. equina* morphologically.

Filaria lachrymalis, Gurlt, 1831. Syn.: (?) *Thelazia Rhodesii*, De Blainville, 1828; *F. bovis*, Baillet, 1858; *F. palpebrarum*, Baillet, 1858.—Found in the lachrymal duct of cattle, whence it may wander into the conjunctival sac. When numerous they may cause irritation or corneal ulceration. The parasite may be directly removed or washed out by injection of an anthelmintic.

Filaria capræ, von Linstow, 1883.—Found by Fedschenko in Turkestan in the tongue muscles of a goat, only the fragments of a female having been examined.

Filaria acutiuscula, Molin, 1857.—Found by Natterer in Brazil in the viscera of two species of peccary (*Dicotyles albirostris* and *D. torquatus*) and beneath the pectoral muscles of *Vulpes azaræ.* Ninni found it under the skin of a dog in Italy.

Filaria Osleri, Cobbold, 1879. Syn.: *Strongylus bronchialis canis*, Osler, 1877.—Found to be the cause of an epizootic broncho-pneumonia in dogs at Montreal by Osler. Rabe and Blumberg seem to have previously observed this worm in small nodules in the mucous membrane of the respiratory passages, each nodule containing several of them. Both males and females were present, the latter being most numerous. The ends of the worms projecting from the nodules presented a villous appearance—ovoviparous.

Filaria oculi canini, Gescheidt, 1833. Syn.: *F. trispinulosa*, Dies., 1851.—Found in the dog's vitreous. Determination doubtful.

Filaria hepatica, Cobbold, 1879.—Nematode larvæ of a doubtful character found by Mather (1843) in Edinburgh in the liver and intestine of a dog which suffered from dyspnœa, vomiting, alternate constipation and diarrhœa, finally bloody vomit, icterus, intense thirst, death. At the autopsy the stomach was found inflamed and exhibited ecchymoses and ulcerations, the duodenum was invaginated, the small intestine was congested and dotted over with small cysts containing filariæ, which were also found free in the bile and hepatic ducts. Blood not examined.

Filaria rytipleurites, Delongchamps, 1824.—Delongchamps discovered this parasite encysted in the adipose tissue of the cockroach (*Periplaneta orientalis*). If the cysts are removed from the insect and placed in a suitable fluid, the worms bore their way out, and may live three or more days in a free condition. Galeb (1878) fed three white rats with infected cockroaches, and, killing them after eight days, found the parasites, which had thrown off their envelopes in the stomachs of the rats, lying in the folds of the mucous membrane. In one rat one male and three females were found, all perfectly developed. Fertilisation takes place in the rat's alimentary canal, and the eggs, escaping with the fæces, are devoured by the cockroaches. The embryos escape from the membranes after they have entered the alimentary canal of the insect, and, boring their way through the intestinal wall, become encysted in the adipose tissue. The rat infects itself by feeding on cockroaches containing the encysted parasites.

Filaria strumosa, Rudolphi.—Found in the mole (*Talpa europœa*) in various parts of Europe. Von Linstow (1887) claimed to have found the larval form encysted in the adipose tissue of a beetle (*Cetonia aurata*).

Filaria uncinata, R.—Occurs in the œsophagus of ducks and geese, causing death at times by its presence. Hamann (1893) discovered that the larvæ develop in *Daphnia pulex* (Rich.), the latter being most plentiful in certain ponds during July-August in Prussia, when the ducks died most. The worms produce tumours which project into the œsophagus, the tumours ranging up to 1 cm. across. The small tumours contained worms 3 mm. long, the large ones worms 18 mm. long. Young ducks were most affected. They seem to die from starvation, being unable to swallow their food on account of the occlusion produced by the tumours. The worms give off embryos, which escape by active motions through the mouth, or more usually with the fæces, the latter being devoured by the Daphnia. The embryos bore their way through the crustacean's intestine, and develop most of the characters of the adult in this host. Sexual maturity is only attained

when the parasite gains access to the duck, which infects itself by feeding on Daphnia.

Filaria Mazzantii, Railliet, 1891.—Only the female worm is known, having being found beneath the skin of the pigeon, the blood of which contained the embryos. Viviparous.

Filaria clava, Wedl, 1856.—Found by Müller in the peritracheal connective tissue of the domestic pigeon.

Filaria Mansoni, Cobbold, 1879.—Found by Manson at Amoy, China, in the eye of the domestic chicken. Magalhaes (1895) found it also in Brazil in chickens and peacocks.

Filaria anatis, Rudolphi, 1809.—Found by Paullinus about the heart of a duck.

BIBLIOGRAPHICAL NOTE.—Besides the filariæ referred to in the text, a large number are further found in mammalia, birds, reptiles, amphibia, fish, mollusca, insects, myriapods, and crustacea, as will be seen by referring to the monograph of Stossisch, who gives a very extensive list of the filariæ (212 !), their hosts, and references to the bibliography. In the above article I have made free use of Railliet's excellent work (adopting his nomenclature), as also of the writings of Manson. In both of these writers' publications will be found references to the literature. A bibliography will be found in a publication of the writer's given below. See also the contributions of British authors of late years in the *British Medical Journal* and *Lancet*, and also refer to the *Centralblatt für Bakteriologie und Parasitenkunde.* It is impossible in the limit of space allowed to include a bibliography of so extensive a subject, but subjoined are some of the more important references to the recent literature ; the rest are to be found in the books of reference or those above mentioned.

LITERATURE.—1. Bancroft, T. L., 1899, *Journ. and Proc. Roy. Soc.* N.S. Wales, xxxiii. ; also *Australasian Med. Gazette.*—2. Charles, R. H., 1892, *Sc. Mem. by Med. Officers of the Army in India*, part vii., Calcutta.—3. Daniels, C. W., 1898, *Brit. Med. Journ.*, i. p. 1011 ; *ibid.*, ii. p. 878 ; *ibid.*, 1899, i. p. 1459.—4. Firket, C., 1895, *Bullet. de l'Acad. de méd. Belge.*—5. Hamann, O., 1893, *Centralbl. f. Bakteriol. u. Parasitenk.*, xiv. 555.—6. De Magalhaes, P. S., 1895, *Bullet. soc. zoolog. France*, xx. 241.—7. Maitland, J., 1897, *Brit. Med. Journ.*, ii. 903.—8. Manson, P., 1894, *ibid.*, i. 1186 ; 1896, *ibid.*, ii. 1379 ; 1897, *ibid.*, ii. p. 1837 ; 1897, Article "Filaria" in Allbutt's *System of Medicine*, London, ii. 1058-1087; 1898, *Tropical Diseases*, London ; 1899, *Brit. Med. Journ.*, ii. 644.—9. Neumann, G., 1895, *Bullet. soc. zoolog. France*, xx. 123.—10. Nuttall, G. H. F., 1899, *Johns Hopkins Hospital Reports*, Baltimore, viii. Nos. 1-2 ; also *Hygienische Rundschau*, Berlin, ix. — 11. Ozzard and Daniels, 1897, *Brit. Guiana Med. Annals.*—12. Plehn, F., 1898, *Die Kamerun-Küste*, etc., Berlin, p. 295.—13. Railliet et Moussu, 1892, *C. r. de la soc. de biolog.*, Paris, (9) iv. 545.—14. Railliet, A., 1895, *Traité de zoologie médicale et agricole*, Paris, 2nd ed., pp. 499-533.—15. Sonsino, P., 1874, *Rendiconto della R. Accad. delle scienze fis. e. mat. di Napoli*, fasc. 6, giugno 1881, *Bulletin de l'Institut Égyptien*, 2e série, No. 2, p. 118 ; 1882, *Med. Times and Gazette*, May ; 1883, *ibid.*, 22, Sept. 29, Oct. 13 ; 1884, *Giornale della R. Accad. di Med. di Torino*, Fasc. 8, agosto 1885, *Bull. de l'Inst. Égyptien*, 2e série, No. 6, p. 146.—16. Stossisch, M., 1897, "Filaria e Spiroptere," *Bollet. d. soc. adriat. di sc. nat. in Trieste*, xviii.—17. Thorpe, V. G., 1896, *Brit. Med. Journ.*, ii. 922.—18. Young, C. W., 1897, *ibid.*, i.

File-cutters' Disease. See Trades, Dangerous (*Lead Poisoning*).

Filicin.—The active principle of the root of *Aspidium filix mas.* See Filix Mas.

Filiform.—Threadlike, *e.g.* the filiform papillæ of the tongue. See Physiology, Digestion (*Mouth*, *Tongue*).

Filix Mas.—The rhizome of *Aspidium Filix mas*, the male fern. It contains an oleo-resin and an active principle, Filicic Acid, which can be isolated as a white amorphous powder. *Preparation* — Extractum Filicis Liquidum. *Dose* — 45 to 90 m. The liquid extract is employed as an anthelmintic for all varieties of tape-worm. It has a disagreeable taste, and on account of its irritating qualities it is often vomited soon after being taken. Doses of from 1-2 ʒ are recommended, given in emulsion with acacia or tragacanth and flavoured with ginger. The prescription should not include water, as this precipitates the oleo-resin. The stomach and intestines must be emptied as far as possible beforehand by fasting and purgatives ; and the dose is to be followed after some hours by another brisk purge to carry away the dead worm. An admirable method of administration is by capsule in doses of 15 m. repeated every half hour until the desired amount has been given. Success cannot be claimed until the head of the worm has been found in the motions. Male fern has been used also with some success for the destruction of *Ankylostoma duodenale.*

Fillet.—The word *fillet* is used in three senses in Medicine : (1) a bandage, such as that put round the arm before the operation of bleeding from a vein was undertaken (to "fill it," *i.e.* the vein) ; (2) an obstetrical snare or bandage to make traction on the fœtus in delayed labours ; (3) a part of the brain. See Physiology, Nervous System, Medulla Oblongata, Structure.

Filling Teeth. See Teeth (*Caries, Treatment*).

Filters. See Water (*Purification*) ; Sewage and Drainage (*Methods, Grain Filters*).

Fimbria.—A fringe or border, *e.g.* the fimbriæ of the abdominal end of the Fallopian tube. See Fallopian Tubes (*Structure*) ; Generation, Female Organs of (*Fallopian Tubes*).

Fingers.

See also Ainhum; Amputations (*Upper Extremity, Hand*); Artificial Limbs (*Hand*); Brain, Physiology (*Functions in Cerebral Cortex, Centres for Fingers*); Bronchi, Bronchiectasis (*Clinical Phenomena, Clubbing of Fingers*); Deformities (*Hand and Fingers*); Fractures (*Phalanges*); Heart, Myocardium and Endocardium (*Effects of Cardiac Disease, Nutritional Changes, "Clubbed Fingers"*); Heart, Congenital Malformations of (*Physical Signs, Clubbing of Fingers*); Lung, Tuberculosis of (*Complications, Integumentary, Clubbing of Fingers*); Nails, Affections of; Raynaud's Disease (*Local Syncope, Fingers*); Sclerodermia.

1. Medical and Surgical Anatomy.—*Tendons* are found mainly in relation to the anterior and posterior aspects. Flexion is performed by the flexor sublimis digitorum, flexor profundus digitorum, and flexor brevis minimi digiti. Extension is produced by the extensor communis digitorum, extensor minimi digiti, and extensor indicis. The fingers are adducted by the palmar interossei, and abducted by the dorsal interossei, and in the case of the little finger by the abductor minimi digiti. The dorsal interossei, too, together with the lumbricales flex the first and extend the second and third phalanges. Lateral incisions into the fingers should if possible be made on the ulnar rather than on the radial aspect, so as to avoid the lumbricales (Kocher). The names of the muscles acting on the thumb indicate the movements produced by them.

As low down as the terminal phalanx in each finger, the flexor tendons are invested by fibrous slips from the palmar fascia, and enclosed too in a synovial sheath which commences opposite the head of the metacarpal bone and extends downwards. In the case of the thumb this sheath is continued up into the forearm, and the tendon sheath of the little finger sometimes communicates with the common flexor sheath. Of the transverse grooves in the skin on the anterior surface, the two distal ones are almost opposite the respective interphalangeal joints, while the most proximal one, which is on a level with the web of the fingers, is about three-quarters of an inch below the metacarpo-phalangeal joint.

Vessels.—The fingers are supplied by the four digital branches of the superficial palmar arch, which are situated laterally beneath the digital nerves, by the dorsalis indicis, radialis indicis, dorsalis pollicis, princeps pollicis, and the two dorsal digital branches of the metacarpal branch of the radial artery.

Nerves.—The flexor sublimis digitorum, the radial half of the flexor profundus digitorum, the abductor pollicis, opponens pollicis, the outer head of the flexor brevis pollicis, and the two radial lumbricales are supplied by the *median nerve.* Sensory fibres from this nerve supply on the anterior aspect the thumb, index, and middle fingers, and radial half of the ring finger; filaments unite too with the dorsal digital branches of the radial nerve to supply the same fingers on the dorsal aspect.

The ulnar half of the flexor profundus digitorum, the muscles of the little finger, the interossei, the two inner lumbricales, the adductor pollicis, and the inner head of the flexor brevis pollicis, are supplied by the *ulnar nerve.* This nerve gives sensory branches to the ulnar side of the little finger and the adjoining sides of the little and ring fingers. It also sends a communicating filament to the branch of the radial nerve supplying the adjacent sides of the ring and middle fingers on the dorsal aspect. The extensor communis digitorum, extensor indicis, extensor minimi digiti, and the extensor muscles of the thumb, are supplied by the posterior interosseous branch of the *musculo-spiral* nerve. The radial branch of the same nerve supplies on the dorsal aspect the thumb, index, and middle fingers, and radial side of the ring finger.

2. Appearances in certain General Diseases, etc.—Much valuable information is to be obtained from a careful study of the fingers. The pursuit of certain occupations may produce distinctive marks. Such are the callosities on the adjacent sides of the index finger and thumb, and between the third and fourth fingers in coachmen, and the callosities on the radial sides of the thumb and forefinger in masons. Owing to the diversity in different individuals of the lines and whorls produced by the papillæ in the skin of the distal phalanges, the study of *finger-prints* is said to furnish most reliable aid towards the identification of persons. Indications of various morbid processes may be detected by an examination of the fingers. The *pulsation* of the *digital vessels* may be unduly forcible on palpation, or even visible, in cases of aortic incompetence, and in other conditions where the peripheral vessels are in a state of undue relaxation. Accompanying this there is often visible capillary pulsation which will be most evident in the finger-nails. *Cyanosis*, whatever be its cause, is often noticeable in the fingers. It is specially pronounced in cases of cardiac disease, whether congenital or otherwise, and of pulmonary emphysema. The cyanosis may, however, be localised, and be a manifestation of Raynaud's disease. This is usually a symmetrical affection; the cyanosis is preceded by local syncope, and is sometimes followed by sloughing and even gangrene of the affected parts. *Clubbed fingers*, or Hippocratic fingers, are common. The terminal phalanges become broadened, the curvature of the nails is increased, and the digits are frequently cyanosed. Such fingers are most often seen in the course of some chronic pulmonary disease, such as phthisis, bronchiectasis, emphysema, or empyema.

but have occasionally been found associated with disease in other organs, for instance with aneurysm of the aorta. "Clubbing" of the fingers must be differentiated from the abnormal state of the digits occurring in acromegaly and in pulmonary osteo-arthropathy. In *acromegaly* the fingers are uniformly enlarged. They are broad too, but there is neither clubbing nor tapering of the finger-tips. The interphalangeal creases are very evident, and the nails which are not disproportionately enlarged are flat and often show longitudinal furrowing. The individual will, in addition, present other features so characteristic of the disease. In *pulmonary osteo-arthropathy* the enlargement, though involving the bones of the fingers, hands, feet, and lower parts of the legs and forearm, is not so uniform as in acromegaly, and the skull is not affected. The terminal phalanges are most markedly involved, being large and broad, the fingers thus somewhat resembling ordinary clubbed fingers. The nails are of large size, very convex, and often present marked longitudinal striation. The diagnosis rests mainly on the presence of some causal pulmonary disease, and on the fact that the enlargement is not confined to the finger-tips alone.

3. Deformities and Distortions. — *Congenital Deformities*, *Dupuytren's contraction*, *spring finger and mallet finger*, *vide* article "Deformities."

Contraction and stiffness of the fingers may result from contraction or section of the flexor or extensor tendons. It may occur, too, after burns or septic inflammation, and in the latter case the worst forms of contraction are due to inflammatory adhesion of the tendons to their synovial sheaths. These conditions are very difficult to treat satisfactorily, but the contraction may be improved if passive movements of the affected digits be continued for sufficient length of time. If there be contraction of the tendons themselves, division may effect considerable improvement; and if the tendons have been divided, union of the cut ends is indicated.

Due to Affections of the Nervous System.—The involuntary, irregular jerky movements of *chorea*, and various *tremors*, *e.g.* paralysis agitans, are usually well seen in the fingers. The movements of *Jacksonian* and of *post-hemiplegic epilepsy* may be confined to the fingers or thumb. *Athetoid movements*, too, are often observed in the fingers, following especially cerebral hemiplegia or diplegia in children. In *epilepsy* the aura may be referred to the fingers, and in the tonic spasms of epilepsy and of tetanus the fingers are tightly clenched in the palm of the hand. Tonic bilateral contractions of the fingers are also seen in *tetany*. In this condition the fingers are flexed at the metacarpo-phalangeal joints and extended at the other joints. The thumb lies in the palm, is rarely bent, but is usually straight with its terminal phalanx pressed against the palmar surface of the second, third, or fourth finger. In rare instances the fingers are clenched over the thumb. The fingers have been likened to those of the obstetrician when he is about to introduce the hand into the vagina. The spasms, though often first manifested in the fingers and toes, are, of course, not always confined to these parts; the arms, legs, etc., may be affected too. These tetanoid spasms are remittent. At first they may persist for only a few hours, while the remissions may last for days or even weeks. The periods of remission are gradually shortened, and each succeeding attack persists longer, until the duration of each paroxysm may be measured by days or weeks.

Spasms or cramps may also be manifestations of an *occupation neurosis*, *e.g.* writer's cramp, or of hysteria, both of which conditions may also cause paresis. They are usually unilateral.

Neuritis or trauma producing *paralysis of the median nerve.*—The effect of this on the fingers is as follows:—The second phalanges cannot be flexed, nor can the distal phalanges of the first and second fingers. The thumb cannot be opposed to the fingers, and there is wasting of the thumb muscles. Sensation is affected in the index, middle, and radial half of the ring finger and the palmar surface of the thumb.

In *paralysis of the ulnar nerve* there is inability to flex the first and to extend the second and third phalanges. The fingers cannot be abducted or adducted, nor can the thumb be adducted. Anteriorly there is loss of sensation over the little and half the ring finger, posteriorly over the little, ring, and half the middle finger. In advanced cases the position of the fingers somewhat resembles that seen in progressive muscular atrophy.

Paralysis of the Musculo-spiral Nerve.—The first phalanges of the fingers and thumb are flexed and cannot be extended. Sensation may be diminished in the region supplied by the nerve.

Paralysis of the extensors should also be noted as occurring in *plumbism*. In advanced cases of *progressive muscular atrophy*, subsequent to the wasting of the thenar and hypothenar eminences, the first phalanges are hyperextended and the more distal phalanges are flexed, the condition being the result of wasting of the interossei and lumbricales, and contraction of the extensors and flexors of the fingers.

4. Injuries.—Detailed remarks regarding injuries of the fingers may be omitted, but attention must be drawn to the important fact that treatment of bruised or wounded fingers should aim at the preservation of as much tissue as possible. *Gangrene* may ensue as a result of bruises and other injuries, as well as after inflammation, arterial obstruction, etc. *Foreign bodies* lodging in the digits may be very troublesome, both from the pain they produce

and the difficulty the surgeon often experiences in removing them.

Fractures, *vide* article "Fractures."

Simple Dislocations at the metacarpo-phalangeal joints are fairly common, the first phalanges being as a rule dislocated *backwards*. The injury occurs from a fall or a blow, and is most frequently seen in the thumb. In this instance the articular surface of the first phalanx lies on the dorsal surface of the meta-carpal bone, and the head of the latter can be felt on the palmar surface. The first phalanx is over-extended, the second phalanx being some-what flexed. The dislocation is often exceedingly difficult to reduce owing to the head of the metacarpal bone being caught between the two divisions of the flexor brevis pollicis. Reduction is facilitated if the patient be anæsthetised, and is effected by first extending the first phalanx, possibly with the aid of a clove hitch or an "Indian puzzle," to a right angle with the meta-carpal, then exerting traction in the same line, while simultaneous pressure is put on the head of the metacarpal bone from the dorsal aspect, and by finally flexing the first phalanx. If in spite of these manipulations the dislocation remains unreduced, the obstructing structures must be divided by the tenotomy knife, and reduction is then easy. To obviate a recurrence of the dislocation the thumb should be kept flexed into the palm for some time afterwards.

Simple dislocations at the interphalangeal joints are not very common, partial dislocation of the middle phalanx being the most frequent. Reduction is as a rule easily performed, but there is often subsequent stiffness of the joint.

Compound dislocations, whether at the inter-phalangeal or metacarpo-phalangeal joints, are also met with. Reduction is as a rule easy, and the treatment otherwise is to be carried out on general surgical principles.

5. Inflammatory Affections.—The fingers may be the seat of ulcers, chancres, bursitis, ganglia, etc., none of which require detailed description here.

Whitlow or paronychia is the term signifying inflammation of a finger. It is almost invariably the result of trauma, which may, however, have been so trivial an abrasion, scratch, or pin-prick that it was disregarded, but which sufficed for the entrance of pathogenic micro-organisms into the tissues. The bacteria most commonly associated with these whitlows are the Staphylo-coccus aureus, S. albus, and streptococci. The state of the general health is an important etiological factor, whitlow occurring, *cæteris paribus*, most frequently in debilitated subjects and in delicate children. Apart from acute periostitis, the inflammation mainly affects either the skin and cellular tissue, or the synovial sheaths and tendons. There are thus two chief varieties of whitlow: (1) *Superficial*, (2) *Deep*.

(1) The former, where chiefly the areolar tissue and skin are involved, is both the more frequent and also the simpler of the two varieties. The inflammation commonly com-mences in one of the terminal phalanges, usually on the palmar surface, and varies greatly in intensity. If the skin alone be affected there may be merely redness and slight pain, or there may also be serous or purulent effusion, manifested as a bulla on the surface of the digit. If the cellular tissues are involved, the signs are more marked. There is greater pain and throbbing, the finger is hot and swollen, and if suppuration has occurred fluctuation may be elicited. If the condition has been neglected the pus may have made its way through the cutis, the inflammation may have involved the tendon sheaths, or if the whitlow commenced at the finger-tip there is a special liability to necrosis of the long flexor tendon and of the terminal phalanx. This variety of whitlow is not usually, unless neglected, of a serious nature, although often accompanied by such general constitutional disturbance as occurs from in-flammatory processes elsewhere. One special form of these whitlows may be mentioned, viz. perionychia. The suppuration here affects the matrix or the root of the nail and causes great pain; the nail becomes detached, or if not, it will usually require to be removed before a cure can be effected.

(2) *The second variety*, in which the tendon sheaths are affected. Here the inflammation starts either in the sheaths themselves or extends to them from the cellular tissue or the bone. The condition is a more serious one, the pain and swelling are more intense, and the con-stitutional symptoms are more severe. The tendons are liable to necrose, and in this variety of whitlow, as well as in the former, the in-flammation may extend to the periosteum and bone and cause necrosis. The anatomical arrangement of the flexor sheaths of the thumb and little finger affords the explanation why this variety of whitlow is more dangerous in the thumb or little finger than in the other digits, and why the suppuration more often extends to the forearm.

Treatment.—In the milder forms, where the skin is chiefly affected, simple wet dressings, preferably of a weak antiseptic nature, may suffice to relieve the pain, and a cure may often result without further treatment. If suppura-tion has occurred with the formation of a bulla, this should be opened, the sero-purulent fluid evacuated, dead epithelium removed, and a wet dressing applied. When the areolar tissues are involved, and even though suppuration has not yet occurred, a free incision is indicated. This is of great benefit in checking any further extension of the suppuration, and in warding off necrosis of the bone and tendons. If there are signs of suppuration in the synovial sheath the incision must be carried down so as to open the

sheath freely, and if the suppuration has spread to the forearm incisions there are also required. Subsequent elevation of the limb and antiseptic wet dressings are necessary until suppuration has ceased, when a dry dressing should be substituted. A purgative should be given at the outset, and the general health attended to by suitable tonic and hygienic treatment. Amputation of the finger may be the proper treatment in some cases, especially when the bone is necrosed, or if the destruction of tendons and other soft parts is so great as to afford little hope of the finger being afterwards in any way useful to the individual.

In some cases the inflammation is extremely intense and spreads rapidly up to the forearm. The condition is really that of a streptococcal cellulitis, and must be treated by free incisions and by continual immersion of the limb in a warm bath of some weak antiseptic. Stimulants should be given internally, antistreptococcic serum is of benefit in some cases, but amputation of the arm may be required to save the patient's life.

6. Tumours of all varieties, simple and malignant, may occur in the fingers, affecting the bones or the soft parts. The most common are *chondromata*, which consist of hyaline cartilage encapsuled in osseous tissue. They usually originate in the neighbourhood of the epiphyseal lines, are elastic and firm unless mucoid degeneration has occurred, painless, as a rule multiple, though sometimes solitary, and at times undergo ossification. They usually arise from the fingers, hands, or feet of children or young adults, and owing to their number, or to the inconvenience they cause, they may require removal. In most cases the best method of removal is by incision of the bony capsule and enucleation of the tumour. Excision of the bone from which they arise, or amputation of the affected fingers, may possibly be advisable if the chondromata are very numerous, or of a mixed sarcomatous nature.

7. Diseases of Bones and Joints.—Acute periostitis and osteomyelitis occur in the fingers as elsewhere. Both conditions may arise from extension to the bone of an ordinary whitlow, and result in partial or more extensive necrosis.

Tuberculous Dactylitis.—This disease is of the same pathological nature as tuberculous disease of other bones. It is of fairly common occurrence in children. The finger is swollen and occasionally painful. The skin, which at an early stage is quite healthy, often, however, presents a red and shiny appearance, with later on evidences of ulceration, sinuses, etc. The necessary treatment varies in different cases. Scraping and excision of the diseased parts may be sufficient, though amputation is often required. In children the condition closely resembles *syphilitic dactylitis.* The latter, which is much less common, occurs as a result of either inherited or acquired syphilis. It is essentially a gummatous periostitis, it may result in necrosis of the bone, and the patient often exhibits other signs of syphilis. *Rachitic thickenings* of the ends of the fingers have been observed in severe cases of rickets.

Acute synovitis, which is as a rule due to injury, simple chronic synovitis, resulting often in some degree of anchylosis and tuberculous disease of the joints, all occur in the fingers as in other joints. *Rheumatism* in all its forms frequently attacks the fingers. As one of the sequelæ of the acute form there may be an extreme degree of ulnar flexion of the fingers. In the ordinary chronic type the finger-joints are often fusiformly enlarged, and there is more or less fibrous anchylosis with stiffness of the joints, and pain and creaking on movement.

Arthritis deformans.—Rheumatoid arthritis in young females often first affects the fingers. It produces a variable degree of swelling about the joints, over which the skin feels hot and is reddened. There is often pain on movement of the fingers. As the disease progresses one sees well-marked muscular wasting, especially of the interossei, with resulting deformity of the fingers. In most cases grating can be made out owing to bony erosion, and in late stages one finds bony anchylosis.

In *gout* the fingers are affected by occasional premonitory painful twinges, by true attacks of gouty "inflammation," and finally by swelling of the joints, deformity of the digits, and by the presence of tophi over which the skin often ulcerates.

Finsen Light. *See* Skin, Tuberculosis (*Treatment, Phototherapy*); Tumours, Inoperable, Treatment (*Phototherapy*); X-Rays and Finsen Light.

Firearms. *See* Medicine, Forensic (*Wounds from Firearms*).

Fires. *See* Ventilation and Warming (*Open Fires*).

First Aid. *See also* Bandages; Fractures; Joints; etc. — A full account of the theory and practice of first aid is outside the scope of this work, but will be found in the references under literature. The object of the present article is to supply such information as will be found valuable in the various emergency cases which come under the treatment of practitioners.

The subject will be discussed in the following order:—

(i.) Shock, concussion, and compression, asphyxia following injury or various forms of poisoning.

(ii.) Improvised antiseptics, dressings, bandages, and splints available for wounds, fractures, sprains, and dislocations.

(iii.) Means of transport.

(iv.) Unconsciousness, epilepsy, apoplexy, hysteria, fainting, various poisons.

(v.) Burns, frost-bite, bites of animals, hæmorrhage, foreign bodies.

(i.) Shock, etc., and its Treatment.—*Shock* results from injury to large surfaces or from the implication of important organs in crushes or buffer accidents; sometimes there is marked shock, mental in character, following mild assaults. In railway accidents and in intoxicated persons the onset of shock may be delayed some hours. The chief symptom of marked cases is great depression of all the vital powers, and constant attention is needed to produce reaction; rest and warmth are to be combined with vigorous chafing of the limbs with hot cloths, raw brandy sponged round the mouth, and teaspoonfuls of hot stimulants given with caution lest sickness be produced.

In *concussion* unconsciousness is, as a rule, of short duration, and continuance of it longer than some minutes would suggest the possibility of more serious injury to the substance of the brain. A blow on the chin, falls on the buttocks or feet when the spine is straight and rigid, are the common indirect causes of concussion. However slight the concussion may be, care should be taken in the treatment; the body warmth is to be maintained, and absolute rest to all the senses; alcohol, except in very severe cases, should be avoided, and later when reaction sets in a dose of opening medicine is advisable.

This condition of concussion may pass into *compression*; frequently, however, there is recovery from unconsciousness, and then a gradual relapse with motor paralysis, the bladder requiring to be emptied. When doubt exists as to the diagnosis from sunstroke, exposure to cold, apoplexy, or neurotic poisoning, the patient should be carefully watched before active interference is undertaken, the treatment in the meantime being the same as for concussion.

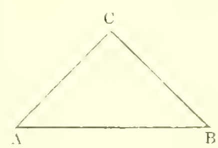
Asphyxia, due to mechanical interference with the oxygenation of the blood, occurs in strangulation or hanging, foreign bodies in the air-passages, œdema following scalds, wasp stings, and corrosive poisons; in addition it is seen in drowning, and where there has been exposure to gases either incompatible with life or poisonous in their nature, as carbonic oxide. The treatment in all such cases is to remove the cause as far as possible, and adopt artificial respiration, which should be continued as long as there is a suspicion of the heart beating. It does not always follow that recovery takes place after the re-establishment of respiration; all such cases should then be carefully watched. Foreign bodies about the air-passages require prompt treatment, the finger should be swept over the back of the throat, and this failing, the trachea must be opened with whatever sharp knife comes to hand, a hairpin will keep the lips of the wound apart while artificial respiration, if needed, is carried out. If blood be entering the trachea the head should be turned well to the side.

The œdema which follows scalds, wasp stings, and corrosive poison comes on very rapidly. If there is time the inhalation of steam should be tried, and failing this, together with indrawing of the chest with inspiration, the trachea must be opened.

The treatment of the apparently drowned is to remove weeds or other foreign bodies from the throat, then to empty the air-passages of water by squeezing the chest and inversion, finally carrying out artificial respiration.

When poisonous gases overcome an individual, as is seen in coal mines, sewers, near limekilns, etc., removal to fresh air is the first treatment, and then the carrying out of artificial respiration. In all cases marked cyanosis not tending to disappear should be treated by the opening of a vein and the withdrawal of blood which is embarrassing the right side of the heart. (*See* "Asphyxia.")

(ii.) Improvised Dressings and Splints.—All wounds should be washed with some *emergency antiseptic*, the most readily obtained being turpentine and alcohol, the latter used diluted with an equal quantity of water; cinders and other foreign bodies should be removed as far as possible and a *dressing* applied. Such dressing should consist of a piece of clean linen dipped in one of the emergency antiseptics, or when the wound is an incised one a piece of tinfoil or waterproof may be applied and kept in place by means of some improvised bandage, the *triangular bandage* being more easily obtained and applied than the roller. It is made as follows:—A piece of cloth, some two to three feet square, is divided diagonally to form two triangles; the chief points in each triangle are the base AB, and the apex C. As a triangle it is used to cover in large areas and to make slings, but when smaller surfaces are to be bandaged the triangle must be folded, the apex C to the base line AB, and the area left once again folded towards the base line; the result is known as the *broad bandage*; folding this broad bandage once again towards the base line makes the *narrow bandage*. In bandaging the *head* for large scalp wounds the open triangle is used; its apex placed over the root of the nose, laterally, or even the occiput, and the base line AB, which lies on the opposite side, is then carried round the head, keeping well below the occiput and just above the eyebrows; it is then knotted and the apex pinned over or included in the knot. Wounds in the region of the *eye* are covered by the narrow bandage, which encircles and passes obliquely round the head. When the



lower jaw is fractured or dislocated the narrow bandage is also employed; the centre includes the chin, and one end is carried up till just in front and above the ear; the other end is carried up in front of the other ear, over the vertex, and down till it meets the first end; here they are twisted on each other so that the direction of the ends now pass one forwards above the eyebrows, the other backwards below the occiput; they are then knotted. This arrangement is also used for retaining dressings in the temporal region or compressing the temporal artery in hæmorrhage from it or its branches.

In burns of the *chest*, dressings can be fixed on one side, for example, the right side, by employing the triangular bandage; the apex is laid well over the shoulder of the same side, the area of the triangle hanging down in front; the base is now carried round the chest to the back and knotted beneath the right shoulder, consequently one end of the base is left longer after the knotting; this end is in turn knotted to the apex. The process is reversed when the back of the chest is to be bandaged.

It is not often that the *shoulder* requires to be bandaged, gunshot, lacerated wounds, and burns being the conditions requiring dressings. Two bandages are needed; one used in the triangular form is placed over the shoulder, the apex lying on the corresponding side of the neck, and the base over the upper arm; the base is now carried round the upper arm and knotted. As in injuries of the upper extremity a sling is advisable, the second bandage is utilised as such, and passes round the neck over the apex of the first bandage which is pinned to it.

As the full-sized triangle is too large for the *elbow* the base is folded towards the apex, so as not only to reduce the size, but to obtain the advantage of the narrow bandage. The apex is placed well above the top of the elbow, and the ends of the base are carried from behind forwards and upwards so as to cross in front of the joint, and then pass backwards, including the apex in this turn; if there is sufficient bandage the ends are carried forward again and knotted in front.

The narrow bandage is used for the *upper arm*, *forearm*, and *palm*; especially in the lower third of the forearm it is useful, cuts being common here, and in such injuries about the wrist investigation should be made concerning the tendons and nerves which may possibly be divided. The palm is often lacerated by falls on it, and cuts involving the palmar arches require localised pressure; in both these conditions the narrow bandage is applied by placing the centre on the palm, the ends pass outwards and inwards, then upwards over the dorsum, where they are crossed and continued round the wrist and knotted.

Smashes of the hand and fingers can be covered by using the whole triangle; the hand is laid on it, the fingers towards the apex, and arranged so that when the apex is turned back it will not only cover the back of the hand, but lie well upon the wrist; the base is now carried round the wrist, enclosing the apex, and they are all knotted together. In wounds or burns about the *hip* two bandages are required; one, employed as a triangle, is placed with the apex on the abdomen, while the base is carried round the thigh and knotted; the other bandage is used as a waist belt, including the apex of the triangle which is knotted to it.

The *thigh*, *knee*, *leg*, and *foot* are all bandaged in a manner similar to that of the upper extremity. (For roller bandages see "Bandages," vol. i.)

Improvised *splints* are of all sorts; among the most suitable and obtainable are wooden boards, leather belting, cardboard, folded newspapers, straw bottle-holders, basket work, heather tied in bundles. In the lower extremity, in addition, longer forms of splints are required, such as broom-handles, unloaded guns, umbrellas, etc. These can be padded with whatever soft material there is at hand,—moss, heather, hay, shavings, feathers, stockings, and in cases where there is no appearance of blood through the clothes they can be left on.

Fractures of the upper extremity do not of necessity require emergency splints, but these give such relief that they should be applied. In the common fracture, that of the collar-bone, when triangular handkerchiefs are not available, the following method is useful:—A walking-stick is placed horizontally behind the back about the level of the scapulæ, both elbows are hooked behind it, and a handkerchief tied to the wrist of the affected arm is carried over the sound shoulder down the back and fastened to the walking-stick. Cardboard, newspapers, and straw are very useful in the treatment of fractures, both of the upper and lower arms; where both forearms are broken, the arms should be crossed and fastened to the same board placed anteriorly. In addition to the short splints, such long splints as have been mentioned are of service in fractures about the thigh, and it must not be forgotten that the sound leg makes also an excellent splint. Fractures of the fibula can be well fixed with an external splint, or should the injured person be wearing riding-boots they can be left on.

The commonest injuries to joints are *sprains*, the wrist, ankle, and knee being chiefly affected; they can be distinguished from dislocation by the movements of the joints being natural; much depends on the first treatment; elastic pressure is the best, obtained by padding the part well with stockings or shawls, wool, etc., and finally bandaging them in place.

Dislocations are best left alone except to support the part beyond, as skilled assistance is usually required to bring about reduction; how-

ever, in dislocations of the phalanges, as happens when a cricket ball strikes the tip of the finger, reduction can be effected by pulling on the distal portion.

(iii.) Means of Transport.—The transport of the injured depends greatly on the amount of assistance the patient himself can give; if he is able to walk the injured person places his sound arm round the assistant's neck, while the latter places his arm round the patient's waist. When walking is impossible, two people can carry an individual by means of the *two-handed seat.* Facing each other, the two assistants grasp right and left hands low down, while the disengaged arms are placed on each other's waist to form a back. The *three-handed seat* is made by an assistant A grasping one of his own wrists, the second assistant B now seizes with one hand A's only free wrist, while A's disengaged hand grasps B's wrist; a back is formed by the second assistant B placing his free arm on A's shoulder. The *four-handed seat* is made by each assistant grasping one of his wrists, *e.g.* the left with the right hand, their disengaged hands now grasp each other's free wrists; the patient here must be able to help by placing his arms round the assistants' necks.

Stretchers can be made from doors, planking, poles interlaced with rope, unloaded guns, or poles pushed through the mouth of a sack and out at the two bottom corners. Coats, especially overcoats, can be utilised by turning the sleeves inside out; the coat is then buttoned in the ordinary way; poles or guns are now pushed through the sleeves. All stretchers should be tested before using them.

The method of lifting the patient on to the stretcher depends on the number of assistants; for instance, if there are three, A, B, and C, A kneels on one knee by the injured side, and if it is a fracture of the lower extremity he devotes his attention to it. B and C kneel on their right knees on the other side and place their arms beneath the patient's buttocks, back, and shoulders; at a given signal all lift together and lay the patient across their left thighs; pausing for a moment, A, B, and C then assume the erect posture and carry the patient to the stretcher, where the performance is reversed. The stretcher should be carried with short steps and the knees slightly bent. In the conveyance of a patient by rail two or three boards should be placed across the seats of a carriage and the stretcher laid on them, assistants sitting in between to steady the injured person.

(iv.) Unconsciousness, Fits, etc.—When a person is discovered in an *unconscious* or *semi-conscious* condition the first thing that must be done is to loosen the clothing about the neck, and to maintain the body warmth, then investigation of the cause may be proceeded with.

Fainting, with its attendant languor, pallor, and feeble pulse, should be treated by keeping the head low, with external and internal stimulation, provided the bleeding point, if there be one, is secured.

Epileptic fits are generally characteristic, a warning cry followed by a fall and rigidity; this condition is followed by clonic convulsions with frothing at the mouth, often blood-stained; then comes a lethargic sleep, from which it is difficult to rouse the patient. If such a case be seen at the onset of the clonic convulsions something should be placed between the teeth so as to prevent the tongue being lacerated; care must be taken not to knock the teeth out. When the convulsive stage has passed off an endeavour should be made to obtain the patient's name and address, as a lucid interval occurs in some cases at this time. "*Fits*" in children, though alarming, do not of necessity imply great seriousness, as they are frequently reflex to some peripheral irritation.

Hysterical fits may resemble epileptic, but there is not the profound sequence of events; the patient is not hurt by the fall, and the conjunctivæ are sensitive. Cold water liberally applied is a most useful restorative.

Apoplexy occurs in the elderly: the sudden onset, the vacant face, stertorous breathing through frothy lips, the unequal pupils with insensitive conjunctivæ, and the paralytic condition of one side, are as a rule sufficient evidence of the ailment. It may resemble alcoholic poisoning, but is more profound, the unilateral paralysis and unequal pupils assisting the diagnosis of the graver affection.

Uræmic fits may resemble epileptic or apoplectic, and if the person so affected be known to suffer from kidney disease, an endeavour should be made to induce perspiration by the application of numerous hot bottles. In uræmic coma the pupils are equal, and the loud hissing character of the breathing assists in distinguishing it from apoplexy.

Excessive moist heat is the common cause of *sunstroke*, the symptoms taking the form either of shock or of sudden unconsciousness, characterised by a feeble pulse, cold skin, with rapidly failing respiration and circulation; the prompt treatment of this condition by free douching with cold water often restores the patient.

Among the poisons which produce unconsciousness *opium* is the commonest; the characteristic symptoms of pin-point pupils, stertorous breathing, and intense drowsiness are generally present when aid is summoned. If seen early emetics may be given, such as mustard and hot water, a teaspoonful to the tumbler repeated every ten minutes; it is better, however, to wash out the stomach if a tube can be obtained, then potassium permanganate can be given, 8 or 10 grains, and the patient kept moving; if this is impossible he can be roused somewhat

by flicking the soles of the feet with a wet towel.

Prussic acid causes almost immediate death, but if seen sufficiently early, rapidly carried out artificial respiration may restore life.

In poisoning by *belladonna*, the symptoms of dilated pupils, thirst, and delirium, with the history, possibly, of eating belladonna berries, is generally conclusive; such a case should be treated by the stomach-pump, the administration of hot coffee, and artificial respiration if needed.

Carbolic acid acts not only as a corrosive, but also as a narcotic; the smell is generally diagnostic; white of egg or Epsom salts should be administered, stimulants also if indicated.

The *corrosive poisons* are chiefly local in action, and require therefore local treatment; after the acid or alkali has been neutralised, olive oil is a useful substance to apply to the mouth and throat.

Oxalic acid acts not only as a corrosive, but frequently causes death by its action as a cardiac depressant; whiting, chalk, plaster, and lime water are all useful antidotes.

Irritant poisons produce burning pain in the throat and gullet, followed by epigastric pain, and later nausea, vomiting, and purging. Arsenic is the most common of the metallic forms, and should be treated by emetics, salts of iron, milk, and stimulants, and later by some soothing drug, such as bismuth. Cantharides and the various forms of food poisoning are also irritant in character.

(v.) Sundry Accidents.—When the clothes catch fire, the best method of extinguishing the flames is to wrap a rug, blanket, or coat tightly round the person; the *burn* which results is to be treated by carefully removing the clothes so as not to detach the thin skin of the blisters; handkerchiefs or linen smeared with vaseline or other greasy preparation should be applied to the part. As the danger from burns and scalds depends on the surface involved, the person should be put to bed and watched for shock.

Frost-bite is more apt to occur when it is damp, cold, and windy than in still frosty weather, and the serious local manifestations are when the extremities or exposed parts become dead white and insensitive; in the treatment of such a case the danger lies in the production of too sudden a reaction with consequent death of the part. A person affected with frost-bite should be treated in a cold room, the parts rubbed with snow or cold water for many minutes. Gradually the increasing body warmth and circulation is further assisted by the administration of hot drinks before transference to a warmer room is attempted. The general condition of drowsiness seen in cases of long exposure to cold should be combated by active massage and rousing. *Wounds* complicated by the possible entrance of *poisonous matter*, as happens in the bites of rabid animals, snakes, etc., require immediate attention. A tourniquet should be applied to the limb above the wound, and if there be no abrasion about the lips, it should be sucked and bleeding encouraged, while incisions round about aid the flow of blood probably poisoned. Cauterisation of the wound should be carried out with a fusee or anything hot which is at hand. At the end of a couple of hours the tourniquet may be loosened for a few minutes, and this is to be repeated provided severe general symptoms do not develop.

When a wound is complicated by injury to blood-vessels it is as a rule associated with *external hæmorrhage*; the bleeding, whether arterial or venous, can be controlled by pressure applied to the actual spot. In the case of *arterial hæmorrhage*, pressure on the artery alone, by means of either the fingers or tourniquet, also suffices, the last being the least tiring. *Venous* bleeding is controlled by a local pad and bandage, or elevation only, when it comes from a varicose vein of the leg. Hæmorrhage from the *nose* is due sometimes to the rupture of a vessel on the anterior part of the septum, and so can easily be compressed; in other cases the severity of the bleeding may call for more active treatment. Plugging the nostril can be carried out by pushing into the nose a portion of fine linen or silk handkerchief on a narrow penholder; this process, like the finger of a glove, is now stuffed with wool or other emergency packing. Persistent bleeding from the *socket of a tooth* can usually be controlled by packing the cavity with wool soaked in turpentine, and placing above this pads of handkerchief so that the subsequent bandaging together of the jaws will induce pressure on the bleeding spot.

Internal hæmorrhage when it is diagnosed should be treated by absolute rest, and if there be great thirst, whatever is given should be cold.

Foreign bodies are as a rule easily removed from the *eye*, unless beneath the upper lid, when eversion of it is required. Foreign bodies fixed in the *cornea* should not be dealt with at once; it is preferable to insert a drop or two of sweet oil between the lids and bandage the eye, till careful treatment can be carried out with a view to possible complications. Children frequently introduce foreign bodies into their *ears*; these are easily extracted, except rounded bodies, more especially peas, which are apt to swell with the heat and moisture; forcible syringing of the ear with warm water often dislodges them, but failing this no amateur attempts should be allowed lest damage be inflicted to the drum.

In the *nose* foreign bodies may be extracted with less fear of injuring important parts.

Fish. *See* Diet (*Animal Foods, Fish*); Food (*Fish*); Heart, Physiology of (*Comparative Anatomy, Circulation in Fishes*); Invalid Feeding (*Fish*); Snake-Bites and Poisonous Fishes; Toxicology (*Food Stuffs, Fish*).

Fisher's Brain Murmur.—A systolic murmur heard over the anterior fontanelle or temporal region in cases of rickets (craniotabes).

Fissure. *See* Brain, Surgery of (*Topography of Fissures of Rolando, Sylvius, etc.*); Cheek, Fissure of; Mammary Gland, Diseases of (*Affections of the Nipple and Areola*); Puerperium, Pathology (*Affections of the Areola*); Rectum, Diseases of (*Piles, Operations, After-Treatment, Fissure*); Teratology.

Fistula. *See* Abdominal Abscess (*Abscess in Abdominal Wall, Fæcal Fistula*); Cautery (*Bright Red Heat, for Fistulous Openings*); Colotomy (*Indications, Strictures with Fistulæ*); Ear, External, Diseases of (*Malformations of Auricle, Fistula Auris Congenita*); Eck's Fistula; Gall-Bladder and Bile Ducts, Diseases of (*Ulceration and its Sequelæ, Fistula*); Intestines, Surgical Affections of (*Obstruction, Treatment, Fistula after Operation*); Kidney, Surgical Affections of (*Renal Fistula*); Labour, Injuries to the Generative Organs (*Vaginal and Vesico-Vaginal Fistulæ from prolonged Pressure*); Lung, Tuberculosis of (*Complications, Alimentary System, Fistula in Ano*); Peritoneum, Tuberculous Peritonitis (*Chronic, Complications, Fæcal Fistulæ*); Pleura, Affections, Surgical (*Empyema, Broncho-pleural Fistula*); Prostate Gland (*Inflammation, Urinary Fistula*); Puerperium, Pathology (*Affections of Mamma, Fistulæ*); Rectum, Diseases of (*Fistula in Ano, Hæmorrhoids, Complications*); Scrotum and Testicle, Diseases of (*Scrotal Fistulæ*); Thiry's Fistula; Umbilicus, Diseases of (*Fistulæ, Fæcal, Urinary, Biliary*); Urachus (*Fistulæ*).

Fitero. *See* Balneology (*Spain*).

Fits. *See* Convulsions; Eclampsia; Epilepsy; Malingering; etc.

Fitz-James, Colony.—An institution for the treatment of the insane, about 36 miles from Paris (near Clermont), in which a great degree of freedom is allowed (no enclosing walls, etc.).

Fixation.—The operation of attaching floating or displaced organs by means of sutures, as in Nephrorrhaphy, Hysteropexy (Vaginal and Ventral), etc.; the term is also applied to the preparation of tissues for microscopical examination.

Fixed Oils.—The official fixed oils are almond, croton, linseed, cod-liver, castor, and olive oil; they are salts formed from the higher fatty acids (*e.g.* oleic and palmitic) and the base glyceryl (C_3H_5); saponification is the formation of soaps and glycerine by the action on fixed oils of caustic alkalies or metallic oxides; the name "fixed" is given to them because their distillation involves decomposition. *See* Pharmacology; Prescribing; and under the various drugs (Amygdala; Croton Oil; etc.).

Flap. *See* Amputations; etc.

Flat Foot. *See* Deformities (*Flat Foot*).

Flat Pelvis. *See* Labour, Prolonged (*Labour with Flat Pelvis*).

Flatulence. *See* Abdomen, Injuries of (*Operation, After-Treatment*); Abdominal Aneurysm (*Pressure Symptoms*); Angina Pectoris (*Etiology, Distension of Stomach*); Gastro-Intestinal Disorders of Infancy (*Disorders of Digestion, Flatulence and Colic*); Heart, Myocardium and Endocardium (*Symptomatology, Stomach Symptoms*); Indigestion (*Varieties, Flatulent*); Stomach and Duodenum, Diseases of (*General Symptomatology, Flatulence*); Vertigo (*Causes*).

Flatus. *See* Labour, Injuries to the Generative Organs (*Laceration of Perineum, Results, Incontinence of Flatus*).

Flax. *See* Trades, Dangerous (*Flax and Linen*).

Flaxseed. *See* Linum, Linseed.

Flea Bites. *See* Petechiæ (*Diagnosis*).

Fleabane.—The oil of Erigeron; it is a yellowish volatile oil, with a taste resembling that of turpentine, obtained from the flowering herb *Erigeron canadense.* It is given in doses of from 10 to 30 m., in capsule, in cases of moderate hæmorrhage from the nose, uterus, etc. It has also been recommended, in combination with other drugs, in the later stages of gonorrhœa.

Fleckfieber. *See* Typhus Fever.

Flesh. *See* Cutis Anserina (*Goose Flesh*); Diet (*Animal Foods*); Food (*Meat*); Physiology, Food and Digestion (*Flesh*); Invalid Feeding; etc.

Flexibilitas Cerea.—The semi-rigidity or wax-like flexibility of the muscles in catalepsy. *See* Catalepsy (*Symptoms*); Hypnotism (*Experimental Phenomena*).

Flexile Collodion.—A mixture of collodion (48 parts), Canada balsam (2 parts), and castor oil (1 part). *See* Collodion.

Flexion. *See* Uterus, Displacements of.

Flexor Response. *See* Babinski's Sign.

Flexure.—A bending or curving of parts or organs of the body, either in the fully-formed state (*e.g.* hepatic flexure of the colon) or in antenatal life (*e.g.* cephalic flexure, neck flexure). *See* Embryology (*Third Week, Dorsal Flexure*).

Flies. *See* Myiasis.

Flint's Murmur.—A murmur heard at the cardiac apex, corresponding to the latter part of diastole, occurring occasionally in cases of aortic incompetence, and probably produced at the mitral orifice.

Flitwick. *See* Balneology (*Great Britain, Chalybeate*); Mineral Waters (*Chalybeate*).

Floating Kidney, Liver, Spleen, etc. *See* Enteroptosis; Kidney, Surgical Affections (*Movable and Floating*); Liver, Diseases of (*Hepatoptosis*); Spleen, Surgery of (*Movable*); Stomach, Surgical Affections of (*Gastroptosis*).

Floccitation or **Floccillation.**—Picking at the bed-clothes, seen in low forms of fever and in delirium tremens; carphology (*q.v.*).

Flocculus. *See* Physiology, Nervous System (*Cerebellum*).

Flooding.—Excessive bleeding, usually from the uterus in connection with labour or in menorrhagia and metrorrhagia from tumours (cancerous or fibroid). *See* Hæmorrhage; Labour; etc.

Florida. *See* Therapeutics (*Health Resorts, American*).

Florida Fever.—A fever often regarded as typhoid or malaria, but possibly distinct from either; also known as "continued thermic fever," "country fever," and "inflammatory fever." *See* Sunstroke.

Flower-Flies. *See* Myiasis.

Flower Rash. *See* Rubella (*Diagnosis*).

Flowers of Sulphur. *See* Sulphur (*Sublimatum*).

Fluctuation. *See* Suppuration; *also* Abdominal Tumours, Diagnosis; Gynecology, Diagnosis in; etc.

Fluids, Examination of Pathological.

See also Ascites (*Characters of Ascitic Fluid*); Aspirator, Uses of; Cryoscopy; Cytodiagnosis; Expectoration; Ovaries, Diseases of (*Ovarian Cysts, Fluid*); etc.

As most of the important pathological fluids are of the nature of transudates and exudates, it is necessary to compare with them the normal lymph transudations that exist in the body. The fluid which percolates through the tissues during life is necessary for their existence. It surrounds the individual cells and is collected in fissures, canals, and sacs of various forms. Of the latter the most important are the large serous sacs, the pleuræ, peritoneum, and those surrounding the central nervous system. These are in communication with lymph-vessels, and these in their turn with blood-vessels. The tissue fluids are derived from the blood, and are used as media not only for the carriage of food constituents from the blood for the different groups of cells, but also for the transmission of substances formed in the tissues to other organs either for the nutrition of the latter or, if the bodies be effete products of metabolism, for excretion. They naturally vary much in composition in different situations. The lymph, as it occurs in the lymphatics, is however characterised by the very high percentage of water, saline taste, and capability of slow coagulation. It is much poorer in proteids than the blood plasma, contains very few white blood corpuscles,

practically no oxygen, and about 50 per cent carbonic acid, which has a lower pressure than in venous, but higher than in arterial blood. Roughly speaking, one may give the following as the average composition of human lymph :—

	Per cent.
Water	95
Total solids	5
(*a*) Proteids	4
(*b*) Fats, cholesterin, and lecithin	0·4
(*c*) Salts	0·6

The amount and character of this fluid may be affected in various ways, *e.g.* by the substances passing in from the alimentary canal, by nutritive or toxic products derived from the tissues, by alterations in blood-pressure, by variations in the composition of the blood, or from alterations in the walls of the blood capillaries. If the fluid differ only slightly from that of the normal lymph, the most marked alteration being that in quantity while the specific gravity remains low, then it is spoken of as a transudate. If it be of higher specific gravity, richer in proteids and cloudy in appearance, then it is termed an exudate. The latter probably arises from an increased permeability of the capillary walls resulting usually from the action of toxic substances formed in the tissues. Whether, in addition to the action on the permeability of the capillary walls, there may not also be an action directly on the secretory activity of the endothelial cells, it is difficult to say. Heidenhain referred to lymphagogues of two descriptions—those which caused a marked increase in lymph production without increase of blood-pressure, the lymph so produced being richer in proteids than before, while there was a diminution in the percentage of these bodies in the blood plasma. Heidenhain believed that this lymph, the so-called "blood-lymph," is produced by the secretory activity of the capillary endothelial cells, while Starling holds that these lymphagogues increase the permeability of the vessel walls, and that the reason for the increase in solids in the lymph is that there is a marked increase in the hepatic lymph which is rich in proteids.

The other class of lymphagogues described by Heidenhain produced a great increase in a very watery lymph, the percentage of water both in the lymph and in the blood being higher than before. Saline solutions are the best examples of this class of lymphagogue. In this case the water is supposed to be principally derived from the tissues, and so Heidenhain termed it "tissue lymph." But here also there seems to be an increased permeability of the capillary walls or, as in the case of the lymphagogue sugar, an increased secretory activity of the endothelial cells. It is not desirable, even if it were possible, to view the three important etiological factors separately, viz. alterations in blood-pressure, composition of blood, and condition of capillary walls. One must remember that the permeability of the capillary walls, and probably also their secretory activity, may be affected by the alterations in blood-pressure as well as by variations in the composition of the blood. When the venous flow is hindered the fluid which passes out is poorer in solid constituents than the normal lymph. It is now known that the blood may be very markedly impoverished, especially with regard to its albuminous constituents, without the production of a transudate. Undoubtedly the most important factor is the alteration in the capillary wall. Such pathological transudates contain very frequently red as well as white blood corpuscles, and often also separated endothelial cells. The specific gravity of transudates varies but slightly, usually from 1·004 to 1·012, and the solid constituents are as a rule less in quantity than in normal lymph. Different pigments may be present in small quantity in transudates, the most important being lutein, oxyhæmoglobin, hæmoglobin, methæmoglobin, hæmatin, and rarely hæmatoporphyrin. These may be identified by spectroscopic examination.

It will be advisable to give at this place a short description of a method which may be employed for a more or less complete analysis of a transudate or exudate, or in fact any other pathological fluid.

(1) General physical characters of the fluid—colour, presence or absence of opalescence, consistence (tested by pouring out a little of the fluid), and deposit.

(2) Specific gravity taken with pyknometer or urinometer.

(3) Reaction.

(4) Deposit. The fluid should be allowed to stand for some time, a small amount of chloroform water being added to prevent decomposition, or better, at once centrifugalised, and the deposit examined microscopically for leucocytes, pus corpuscles, red blood corpuscles, epithelial cells, crystals, etc. The supernatant fluid should be examined spectroscopically.

Chemical Analysis of the Fluid.—(1) *Total Solids.*—Take 20 c.c. in weighed porcelain dish, evaporate to dryness on water bath, then *in vacuo* over sulphuric acid, and finally in hot air bath at 105°-110° C., until of constant weight. Cool over sulphuric acid in exsiccator and weigh. State *total solids* in percentage.

(2) *Glucose, Fats, Lecithin, Cholesterin.*—Extract the dried residue, obtained as described above, with boiling alcohol and ether for one hour on water bath with an upright condenser fitted on to flask in order to prevent loss of fluid. Make up the alcoholic ethereal extract to a fixed volume, say 200 c.c. Divide this into two equal portions; in one examine for glucose, in the other for fats, lecithin, and cholesterin.

(*a*) *Glucose.*—Evaporate down one portion (100 c.c.) to dryness, and then extract residue with hot water in order to dissolve the glucose.

Now estimate the reducing power of this solution before and after fermentation with yeast in order to determine whether any reducing substance apart from fermentable glucose be present. From the amount of Fehling solution reduced the quantity of glucose present can be calculated.

(*b*) *Fats, Cholesterin, Lecithin.*—Evaporate down the other portion at first on the water bath, and then in the exsiccator. Extract residue with warm ether, evaporate off the ether and weigh the residue = fats, cholesterin, lecithin.

To estimate these separately the residue is dissolved in hot alcohol, and then absolute alcohol containing an excess of caustic potash is added. The mixture is kept on the water bath at boiling point for one hour, and the alcohol then removed by evaporation. The residue contains potassium soaps, glycerine, caustic potash, glycero-phosphate of potassium, cholin, and cholesterin. Of these substances only two require to be estimated, namely, the glycero-phosphate of potassium and cholesterin. Dissolve the residue in water and pour the solution into a flask, then extract with an equal quantity of ether, fresh ether being used three or four times. The ethereal layers are poured off and collected, and the ether evaporated off, the *cholesterin* residue then being weighed.

The watery extract is evaporated down to dryness, incinerated with potassium nitrate, the ash dissolved in hot water, and the phosphate estimated.

The amount of lecithin is obtained by multiplying the weight of magnesium pyrophosphate by 7·27.

If the amounts of cholesterin and lecithin be added together, and the sum subtracted from the total weight of the residue of the alcoholic-ethereal extract, the amount of fat is obtained.

(3) *Proteids.*—The most important ones to estimate are those coagulable by heating. Take 25 c.c. of the fluid and dilute with four times its volume of water, neutralise the fluid, bring to boiling point, and then acidify with a few drops of weak acetic acid. Filter, wash precipitate with boiling water, alcohol, and ether, then dry first at moderate and then at higher temperatures up to 115° C. Cool in exsiccator and weigh. Dry until no more weight is lost. Incinerate the dry residue and subtract weight of ash from that of dried coagulum. In this way the coagulable proteids, free from salts, are estimated. The other proteids that may be met with in pathological fluids will be referred to later on in this article.

Composition of Transudates and Exudates.—Not only is the amount of proteid in inflammatory exudates greater than in transudates, but the decomposition products of the albuminous substances also share in the increase. Paijkull made the interesting observation that inflammatory exudates always contain nucleo-albumins, while transudates do not. The amount of proteid in different serous transudates varies, always being greater in pericardial, pleural, and peritoneal fluids than in the arachnoideal. This may be due to a different degree of permeability of the capillary walls in these regions.

The specific gravity of different transudates and exudates runs parallel with the amount of albumin which they contain. The proteids which are most commonly met with are serum albumin, serum globulin, and a small quantity of fibrinogen. Traces of mucin and nucleo-albumin are also often present. Albumoses and peptones never seem to be present, with the possible exception of the cerebro-spinal fluid. Transudates, unlike exudates, if they undergo coagulation at all, only do so very slowly, owing to the small amount of fibrinogen which they contain. The gases are the same as those that occur in lymph. Glucose is practically always present and very often traces of levulose. Frequently other reducing substances have been met with. Traces of sarcolactic, uric, and succinic acids, inosit and creatin also are often to be discovered. The amount of urea varies greatly in different transudates; very often only traces, however, are present. It is still doubtful whether pyrocatechin occur or not. The amount of fat varies greatly just as it does in the lymph, in certain cases when there has been a rupture of lacteals the peritoneal fluid may contain large quantities of fat. Cholesterin and lecithin are usually present in small quantities.

Pericardial Fluid.—One can obtain sufficient of this fluid for examination even under normal conditions. It is of a light yellow tint, of low specific gravity (1·006 to 1·013) and slightly viscid. Probably the most accurate analysis of this fluid is one given by Hammarsten:—

	Per cent.		
Water	96·085		
Solids	3·915		
Proteids	2·86	Fibrin	0·031
		Globulin	0·595
		Albumin	2·234
Soluble salts	0·860		
Insoluble salts	0·015		
Extractives	0·200		

Pathologically this fluid may be simply increased in the amount of the transudation, or it may show an increase in the fibrin. In other cases it may contain blood or pus, or constituents derived from these or chyle. In the latter case the fluid is richer in solids than under other conditions (Hasebroek).

Pleural Fluid.—This varies greatly in con-

sistence and specific gravity. Under normal conditions it is present in such small quantity that it has never been analysed. Pathologically, it may be serous, sero-fibrinous, sero-purulent, or purulent, and hence it varies greatly in specific gravity, owing to the varying amounts of proteid present. In hydrothorax the specific gravity is usually much lower than in pleuritis (as a rule being about 1·01), owing to the fact that the amount of proteid in the former condition is from 1 to 3 per cent, while in the latter about 3 to 7 per cent is present. The specific gravity in pleuritis is very often higher than 1·020. The total solids in some cases of empyema may rise to 10 per cent. The following table gives the results of some analyses made by Halliburton:—

Case.	Sp. Gr.	Total Proteid per cent.	Fibrin.	Ser. Globulin.	Ser. Albumin.
1. Pleurisy (acute)	1·023	5·132	0·016	3·002	2·114
2. ,, ,,	1·020	3·437	0·017	1·240	1·189
3. ,, ,,	1·020	5·201	0·108	1·760	3·330
4. Hydrothorax (Bright's disease)	1·015	2·518	0·006	0·659	1·851
5. ,, ,, ,,	1·012	1·324	0·006	0·402	0·915
6. Hydrothorax (heart disease)	1·016	1·482	0·013	0·779	0·700

Peritoneal Fluid.—Normally the quantity of this fluid is small, but under different pathological conditions it may undergo a very marked increase. If the increase in the fluid be due to the so-called hydræmic condition of the blood, or, more important, if it arise from venous congestion, then the fluid has the typical characters of a transudate, that is to say, it is of low specific gravity, clear and watery in appearance, coagulating only with great difficulty, and almost free from former elements. Under such conditions the amount of proteid varies, as a rule, from about 0·5 to 3 per cent. In inflammatory processes the fluid poured out is characterised especially by the presence of formed elements by its comparatively high specific gravity, and by the fact that it rapidly undergoes coagulation. In appearance it is also different from the transudates, being usually cloudy, and often of a slightly brown tint, owing to admixture with blood pigment. The ascitic fluid may be rich in fat, especially in the so-called chylous ascites due to rupture of the lacteals, the percentage often rising as high as 4·5 per cent. In addition to serum albumin and serum globulin mucin may be present, which can be detected in the filtrate obtained after boiling and acidifying the fluid. This last-mentioned proteid on boiling with weak acids furnishes glucose. If fluid from an ovarian cyst should become mixed with the peritoneal fluid, then instead of, or in addition to, mucin, another closely allied proteid, pseudomucin, may occur in the filtrate. This body is distinguished from mucin by the fact that it is not precipitated by acetic acid. If the fluid be purulent, then the constituents derived from the degenerating pus cells will be present. All or some of the substances referred to as occurring in transudates, lymph, etc., may be met with in the ascitic fluid, *e.g.* urea, uric acid, glucose, etc.

Case.	Sp. Gr.	Proteid percentage.	Same by Reuss' Formula.
A. *Exudates*			
Peritonitis (tuberculous)	1·020	4·753	4·700
,, ,,	1·021	4·770	5·070
B. *Transudates*			
Ascites—			
1. Nephritis parenchym	1·007	0·138	0·012
2. Cirrhosis hepat.	1·013	1·963	2·075
3. ,, ,,	1·014	2·634	2·560

Bernheim gives the above analyses of fluids obtained in different conditions. He estimated the amount of proteids and also calculated the percentage by Reuss' formula from the specific gravity. Reuss' formula is $P = \frac{3}{8}(S - 1000) - 2{\cdot}8$ (P = Proteid per cent, S = Sp. Gravity (reckoned for 1000)).

Cerebro-spinal Fluid.—In chronic hydrocephalus this fluid is of low specific gravity, containing only a small percentage of solids (1·2 to 2 per cent). These include globulin, albumose, rarely albumin, glucose, inorganic salts, and probably pyrocatechin.

Ovarian Cysts.—The *serous cysts* of the Graafian follicles contain a fluid which is of low specific gravity (1·003 to 1·020), and therefore poor in solids. It does not differ from the other serous fluids previously described.

Proliferous Ovarian Cysts.—In the smaller ones the fluid is much thicker than in the larger ones, and is usually more or less colloidal in nature. In some cases it may almost be as firm as a jelly, while in others it is a stringy, viscid fluid. It has usually a bluish white, opalescent appearance, but it is often also yellow or brown in colour, due to the presence of broken-down blood pigment. The reaction is usually alkaline. The specific gravity varies very much, in some cases being as high as 1·050, and in the same way the amount of solids varies. The fluid, as a rule, does not coagulate on standing. The most important constituent is the characteristic proteid, pseudomucin, which possesses, in common with the mucins, a carbohydrate radicle, but, unlike them, it is not precipitated by acetic acid. The substance termed "colloid," which is said to occur in these cystic tumours, is not well

defined, being in all probability a mixture of pseudomucin and other proteids. Serum albumin, serum globulin, mucin, and fibrinogen may be present, but the last mentioned usually only in traces. About 1 per cent salts and 0·5 per cent extractives and fats also occur, and in addition a small quantity of cholesterin. The most important suspended bodies are red and white blood corpuscles, epithelial cells, some having undergone fatty, others colloidal degeneration, crystals of fatty acids, and cholesterin.

The fluid in *parovarian cysts* is of low specific gravity, usually very pale in colour, poor in solids, and apparently never containing pseudomucin.

The constituents of the dermoid cysts are, as one would expect, of the most varied description.

Cysts occurring in other parts are usually characterised by the occurrence of the special constituents of the tissues in which they appear. Thus cysts of the pancreas contain the bodies found in the pancreatic juice, and should always be examined for the presence of enzymes. Thus to one portion of the fluid fibrin should be added, and the mixture kept at body temperature for one or two hours, when it should be tested for albumoses and peptones. To another portion starch should be added, and after half an hour's digestion at body temperature the fluid should be tested for dextrins and maltose. It is not necessary to test for the fat-splitting ferment. Cysts of the kidney should be examined for urea, uric acid, and the different forms of renal epithelium; cysts of the mammary gland for the milk constituents; cysts of the salivary ducts for salivary constituents. There is, however, an absolutely distinct form of cyst that requires to be referred to briefly, viz. *the hydatid cyst.* These vary greatly in size, and hence in the amount and consistence of the fluid which they contain. As a rule the specific gravity is low, 1·004-1·012, and usually only a trace of albumin and a comparatively large proportion of salts, especially sodium chloride (0·3-0·5 per cent), are present. These cysts very often contain crystals of hæmatoidin, and are also frequently stained with decomposed blood pigments. More important than the chemical is the microscopical examination. If the fluid be allowed to stand for some time a granular deposit settles down which should be examined microscopically for the scolices developed from the inner layer of the membrane of the cyst, and also for the ectocyst. The lateral disc-like suckers may be seen with the low or high power, and also the hooklets arranged circularly. These also may be seen in the fluid along with threads of the laminated membrane. The laminæ of which the membrane is composed can be easily separated by teasing when they are seen to be cross striated.

Hydrocele fluid varies in colour, light yellow, brown, or greenish brown. It is of comparatively high specific gravity, 1·015-1·030, and is thus rich in solids (5-8 per cent). It may coagulate spontaneously, but as a rule blood plasma or serum or fibrin ferment requires to be added. Very often cholesterin crystals are present.

Spermatocele fluid is usually thin, colourless, of low specific gravity (1·005-1·010), and it coagulates neither spontaneously nor on the addition of fibrin-ferment. The difference in the composition of this fluid from that of hydrocele is well shown in the following analysis by Hammarsten:—

Constituents.	Hydrocele fluid.	Spermatocele fluid.
Water	93·885	98·683
Solids	6·115	1·217
Fibrin	0·059	...
Globulin	1·325	0·059
Serum albumin	3·594	0·182
Ethereal extract	0·402	1·076
Soluble salts	0·860	
Insoluble salts	0·066	

Blister Fluids, Bullæ of Pemphigus, etc.—In these the fluid is usually of high specific gravity, being rich, the solids often rising to 5 per cent and higher. Cellular elements are often present, the most important being granular leucocytes. The fluid in pemphigus bullæ often contains coarse eosinophilous, polymorpho-nuclear leucocytes, and it is an interesting fact that in such cases, if artificial blisters be raised, they are found to contain not the coarse eosinophilous leucocytes, but the ordinary finely granular oxyphil white blood corpuscles. (*See also* "Gout.")

Synovial fluid may be increased in various pathological conditions. It is characterised by its viscous consistence, alkaline reaction, and presence of degenerated cells. The body which renders the fluid viscid is in some points similar to mucin, while in others it differs, *e.g.* it does not contain a carbohydrate radicle. It is undoubtedly a proteid, and has been termed by Salkowski synovin. The amount of the solids varies greatly, usually being about 3 to 5 per cent. The fluid contained in bursæ is very similar to this.

Pus consists of a fluid, the serum, in which corpuscles float, and its consistence varies with the relative amounts of these constituents. It may be a thin watery fluid or a thick semi-solid mass, and hence the specific gravity is a variable one. The reaction of fresh pus is alkaline, but may in the early stages of decomposition react acid, becoming again alkaline after more prolonged decomposition. The serum of pus is, as a rule, light yellow in colour, with a tinge of green or brown in it. It will not coagulate spontaneously or on the addition of fibrinogen, that is to say, it does not contain fibrin-ferment. Its chief constituents are those of the blood

serum, except that it contains a nucleo-albumin derived from the breaking down of the pus cells. This body, nucleo-histon, is precipitated on the addition of acetic acid to the fluid. The pus corpuscles have the same general composition as the leucocytes, but are characterised by the presence of a nucleo-proteid which swells up in sodium chloride solution. Nuclein is present in the nucleus, while lecithin, cholesterin, glycogen, purin bases, fats and soaps occur in the protoplasm. In an analysis of pus one of the most distinctive points is the large amount of phosphorus present. In old stagnant pus many other decomposition products may appear, *e.g.* leucin, tyrosin, formic, butyric, and valerianic acids. The yellow pigment that occurs in pus is termed pyoxanthose, the blue pigment that occasionally is present pyocyanin.

The Bacteriology of Pathological Fluids

General Procedure.—Whether the fluid be removed during life or after death, and whatever position in the body it may have occupied, the general procedure is the same—(1) to collect a sufficient quantity without contamination; (2) to prepare films for microscopic examination; (3) to isolate pure cultures of the organisms present; and (4) to test their pathogenic effect in suitable animals. Unless each of these particulars has been fully investigated, the research is theoretically inconclusive, and any deduction must be made with reservation; but for many practical purposes it is not essential that the process should be exhaustive; other data, clinical or pathological, may confirm the diagnosis indicated by an incomplete bacteriological examination. Moreover, in some conditions the methods are simple and easy, and the causal bacteria readily identified, whereas in other conditions the process becomes tedious and complex, and the result may remain obscure in spite of every effort. Thus the presence of tubercle bacilli may in most instances with reasonable accuracy be determined by suitable staining and microscopic examination; while in other fluids, such as that obtained by lumbar puncture in suspected tuberculosis meningitis, the utmost care in centrifugalising, in staining and examining films, in cultivation on glycerine media, and in the inoculation of animals, may be barren of result, even though at the autopsy the morbid anatomy is characteristic.

In collecting fluids during life the chief precautions to be observed include the thorough disinfection of the patient's skin at the seat of puncture, the use of an aseptic syringe or aspirator, and the reception of the fluid in a sterile vessel. It is obvious that antiseptic and disinfecting substances should not be employed in such a way as to allow any admixture with the fluid to be examined. In the post-mortem room, also, special precautions must be taken to collect the fluid without contamination.

When the fluid is only slightly turbid a considerable quantity should be taken, several pints if possible, to admit of sedimentation; but when distinctly purulent, a few ounces will be found ample. It may happen that, for various reasons, no fluid is obtained, only some semi-solid material being withdrawn on the point of the needle. From that alone films may be prepared, cultures obtained, and the diagnosis successfully completed, if the observer has taken care to have cover-glasses and culture media ready to hand.

Intrathoracic Effusions, Pleural and Pericardial.—As observations become more extensive, and bacteriological methods more perfect, there is a gradual accumulation of evidence in support of the statement that, with few exceptions, inflammatory exudations into the serous cavities are due to the action of micro-organisms. Pleuritic fluids, being more readily accessible during life than those in any other serous sac, have been studied with most detail and fully bear out this contention. If the exudation be mainly serous, it is strongly held by many observers that the tubercle bacillus is the chief causal agent. But the abundant effusion of fluid and the paucity of the bacilli render their detection a matter of extreme difficulty even in cases whose tuberculous nature is put beyond doubt by clinical or pathological data. This obstacle to a complete bacteriological diagnosis during life occurs in practically all serous effusions due to tubercle bacilli, in spite of the utmost care in centrifugalising the fluid, and in staining and examining films from the sediment. Incubation on glycerinised media usually also fails, though inoculation of animals may succeed where all other means prove unsuccessful. When a tuberculous pleurisy has become purulent, it is usually due to the invasion of some pyogenic microbe in addition to the tubercle bacillus.

Inflammation of the pleura accompanied by suppurative or sero-purulent effusion may be due to one or more of many different kinds of bacteria, but apart from punctured wounds of the chest and traumatic conditions generally, there are two organisms of special importance and frequency in empyema, the streptococcus pyogenes and Fraenkel's pneumococcus. In the experience of the writer the pneumococcus is more common than the streptococcus both in the child and in the adult, even when the pleuritic conditions which are associated with acute lobar pneumonia are excluded. It is noteworthy that many cases of pneumococcal empyema arise without any evidence that they have followed or have accompanied an acute pneumonia. In such cases one often finds that the organisms have undergone degeneration to such an extent that ordinary methods of staining practically do not reveal their typical structure. Cultures prepared from such fluids very often do not

grow, and even inoculation of the rabbit may fail to induce the characteristic septicæmia. In fact, the writer has found that inoculation of the rabbit usually does fail, even when a considerable quantity of pleural pus is injected, whereas inoculation even of a small quantity of an emulsion prepared from pneumonic lung tissue usually succeeds. Yet, where other means have proved futile, the organisms may with certainty be identified by microscopic examination of films specially stained to show the capsules of the degenerating cocci.

The method which has given the writer most satisfaction is that recently devised by Richard Muir and hitherto unpublished. The essential part of the process consists in the use of a special mordant prepared by mixing two parts of a 20 per cent watery solution of tannic acid, two parts of a saturated watery solution of mercuric chloride, and five parts of a saturated watery solution of potash alum. A thin film of the suspected material is spread out on a clean cover-glass, and gently dried over a flame. A few drops of the mordant are filtered on to the film, allowed to remain for about two minutes, and thoroughly washed in water, then in methylated spirit, and in water again. The film is now stained in Ziehl-Neelsen's carbol-fuchsin for two or three minutes, while very gentle heat is applied till the steam begins to rise. After washing in water, the mordant is again applied for about two minutes, and washed off as before. Finally the film is stained for about two minutes in a saturated watery solution of methylene blue, washed in water, differentiated in methylated spirit, dehydrated in absolute alcohol, cleared in xylol, and mounted in balsam.

By this means the bodies of the pneumococci take on a bright red colour, while their capsules are stained blue. When degenerated forms are present, it is found that the capsules persist and are recognisable long after the cocci within have lost all semblance of their normal form, and even after the cocci have disappeared. It is not uncommon to find that several cocci retain their characteristic structure, but that the great majority are in all stages of degeneration, some being represented by minute streaks or dots within the capsule, others by the presence of the capsule alone.

Of the other organisms which may cause empyema the streptococcus has already been mentioned as by far the most common. It usually occurs in a pure condition, and may be recognised and isolated with comparative ease, even when there has occurred a secondary infection by other organisms. Bacteria less commonly present are the staphylococci, the micrococcus tetragenus, and the bacillus coli communis, the last named occurring especially in suppurative pleurisy associated with or following a septic condition of the peritoneum or abdominal organs. Among those still more rarely found in pleural effusion should be mentioned the typhoid bacillus after enteric fever, and the influenza bacillus (Pfeiffer).

Pericardial Effusions.—Much less is known of the bacteriology of pericardial effusions. For obvious reasons the fluid is seldom examined during life unless excessive in amount — an event of infrequent occurrence—or unless suppuration is suspected. Most cases of simple acute pericarditis are, therefore, not investigated. The frequent association of pericarditis with rheumatism and chronic nephritis would appear to indicate that most simple inflammatory exudations in the sac are not etiologically related to any known organism. The tubercle bacillus must, however, be noted as the causal agent in some cases.

Suppurative conditions of the pericardium are much more commonly the result of secondary infection from other foci of suppuration in the body, and it is here necessary only to state that any of the pyogenic organisms, including the pneumococcus, may be found in the pericardial pus. As in the pleura, however, a pneumococcal pericarditis may occur as a primary infection.

Peritoneal Effusions.— Peritoneal inflammatory effusions can almost without exception be correlated with some known microbe. In the common form of acute peritonitis secondary to some lesion of the alimentary tract, whereby infection of the peritoneum occurs from the contents of the bowel, whether actual perforation exists or merely local damage to the wall, the factor of greatest significance is the organism or closely related group of organisms, denoted by the term bacillus coli communis. Sometimes these are the only organisms present, but more often there is a multiple infection by the numerous intestinal bacteria, among which the bacillus coli may or may not predominate. In some cases after perforation the infection may be so mixed, and the organisms all so vigorous in their growth, that it is hard to say which is the causal agent even after experimental inoculation. It has been suggested that an indication may be obtained by examining the blood also, as presumably it would be infected, if at all, by the organism or organisms of highest pathogenicity.

In many cases of septic peritonitis, more or less localised to the pelvis, and occurring especially in women, the gonococcus may be found. It is noteworthy that microscopic examination alone can never afford a satisfactory proof of the presence of gonococci, as their reactions, morphology, and relations to leucocytes may be simulated by other diplococci found in urethral and vesical diseases and in some associated pelvic conditions. It is essential to complete diagnosis that a culture should be prepared on Wertheim's media, or, if none is available, on blood agar. When, however, the staining reactions

and microscopic characters coincide with those of gonococci, the very fact that attempted cultures were unsuccessful may be regarded as further presumption in favour of their identity.

In other peritoneal exudations there may occur streptococci, staphylococci, and even pneumococci, especially when there has been an extension from an intrathoracic focus. Bacilli closely allied to Friedländer's pneumo-bacillus have also been found, and the typhoid bacillus may, though rarely, infect the peritoneum during or after enteric fever. Mention should also be made of actinomycosis and higher fungoid growths as occasionally productive of abdominal inflammations and suppurations.

In chronic peritoneal conditions the tubercle bacillus plays an important part, but here the diagnosis is made from the clinical phenomena or from the morbid anatomy with greater certainty than could be afforded by a search for the bacilli themselves.

Cerebro-spinal Effusions.—An acute or subacute inflammatory effusion from the meninges of the brain or spinal cord is almost invariably due to the invasion of micro-organisms. An examination in life is rarely attempted directly from the intracranial exudate, but valuable information may be obtained by means of lumbar puncture, as in many forms of meningitis the spinal membranes are also affected. It has been suggested that, in cases where permission to examine the head has been refused after death, lumbar puncture may be allowed and the diagnosis thus confirmed.

First in importance and frequency is the tubercle bacillus, but, in the common form of tuberculous cerebro-spinal meningitis in children, the difficulty of finding the bacilli is often very great, even when after death all the cerebro-spinal fluid is collected. In lumbar puncture during life a considerable quantity of fluid is often obtained, but the diagnostic value of this operation is not very great. For in the first place the bacilli are scanty and hard to find, and in the second place, if the microscopic examination of stained films does not reveal the bacilli, the diagnosis is usually completed by the post-mortem report before cultures can grow, or inoculation of animals yield any result.

Next in frequency and importance is the pneumococcus (Fraenkel), which produces, as a rule, a suppurative exudation most commonly over the vertex of the brain, and may or may not be associated with an acute pneumonia. It is important to observe that the pneumococcus must be very carefully identified, not only by its morphological characters and staining reaction (including retention of stain by Gram's method), but also by cultural and experimental data. For other diplococci may occur in the cerebro-spinal exudate, notably the diplococcus intracellularis meningitidis of Weichselbaum, found especially in cases of epidemic cerebro-spinal meningitis, and a diplococcus described by Still in the condition which he designates "simple posterior basic meningitis of infants." In all probability the latter is only an attenuated variety of the former, and the biological points of difference between them are such as would be indicated by corresponding differences in virulence. Morphologically they are indistinguishable, and each in its microscopic characters, in its staining reactions (including loss of stain by Gram's method), and in its relation to leucocytes, closely resembles the gonococcus.

Other organisms which may be found alone or in combination include the pyogenic bacteria, especially in the streptococcus, and also Friedländer's bacillus, the influenza bacillus (Pfeiffer), and other microbes of infrequent occurrence.

The possibility of infection from the naso-pharynx by the ethmoidal air cells and cribriform plate on the one hand, or by the Eustachian tube and middle ear on the other, must always be kept in view, and more particularly when the exudation is distributed over the vertex of the cerebral hemispheres. Of great importance also is the fact that bacteria may reach the cerebral by way of the spinal meninges, and that the latter are especially liable to infection, either from the pleuræ and mediastinal glands, or from the peritoneum and abdominal glands, along the lymphatics that accompany the spinal nerve-roots. The frequency of infection by this channel has for long been pointed out by Greenfield in the ordinary form of tuberculous cerebro-spinal meningitis, and, in all probability, other forms of basal-meningitis arise in a similar way.

Flukes. *See* PARASITES (*Helminths, Trematodes*).

Fluorescine. *See* CORNEA (*Bullous Affections, Dendritic Ulcer, Staining with fluorescine*); EYE, CLINICAL EXAMINATION OF (*Cornea, Diagnosis of abrasion by staining with fluorescine*).

"Flush Area."—The facial area which exhibits acne rosacea in cases of hepatic cirrhosis; it corresponds roughly to that involved in lupus erythematosus. *See* LIVER, DISEASES OF (*Portal Cirrhosis, Signs*).

Flushings. *See* CLIMACTERIC INSANITY; MENOPAUSE (*Disorders*); MENSTRUATION AND ITS DISORDERS (*Menopause*).

"Fluttering." *See* HEART, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology*).

Flux.—A discharge, *e.g.* "bloody flux" or dysentery, menstrual flux or menstruation.

Focus. *See* PHYSIOLOGY, SENSES (*Vision*); ACCOMMODATION; etc.

Fœniculi Fructus. *See* Fennel Fruit.

Fœticide.—The emptying of the pregnant uterus at a date anterior to viability, which necessarily involves death of the fœtus; this, of course, may be brought about as a criminal act or to save the mother's life (therapeutic fœticide).

Fœtus and Ovum, Development of.

See also Abortion (*Causes, etc.*); Achondroplasia; Antenatal Pathology; Ectopic Gestation (*Fœtus*); Embryology; Heart, Congenital Malformations of; Labour, Physiology of (*Passenger, Fœtus*); Labour, Diagnosis and Mechanism (*Fœtus*); Labour, Faults in the Passenger (*Fœtal Malformations, etc.*); Labour, Operations (*Forceps, etc.*); Medicine, Forensic (*Abortion, Infanticide, etc.*); Maternal Impressions (*Fœtal Deformities*); Ovaries, Diseases of (*Dermoid Cysts, Fœtal Inclusions*); Pregnancy, Diagnosis (*Fœtal Signs and Symptoms*); Pregnancy, Multiple; Pregnancy, Pathology (*Affections of Ovum and Decidua*); Pregnancy, Pathology (*Intra-uterine Diseases and Death of Fœtus*); Pregnancy, Hæmorrhage in (*Fœtal Mortality*); Rickets (*Fœtal*); Sclerema Neonatorum (*Fœtus Rigidus*); Syphilis (*Hereditary*); Teratology.

The Graafian Follicle.—Graafian follicles are the essential elements of the ovary, since they contain the ova. Enormous numbers of these follicles exist in the ovaries of new-born female children, a fact known to the anatomist Sappey, who estimated that two women would suffice to populate a city as large as Paris if all their ova were fruitful. At puberty they are much less numerous, as age advances their number diminishes, and from the ovaries of aged persons they are entirely absent. Three different kinds of follicles, which, however, merely represent stages of development, are described: viz. *primary* follicles, *growing* follicles, and *ripened* follicles. The ovaries, at birth, contain as a rule only *primary* follicles. These are invisible to the naked eye, and consist of a single large round cell, the ovum, closely invested with a delicate layer of flattened cells, the follicular epithelium; the ovum consists of a mass of protoplasm, with a single or rarely a double nucleus, and no cell envelope. *Growing* follicles are numerous at puberty. Remarkable changes occur at this period in the follicular cells, while the ovum itself undergoes little alteration. The follicular cells proliferate freely by fission, become cubical in shape, and arranged in several layers around the ovum, thus forming the *stratum granulosum*. Outside this a well-defined connective tissue capsule is formed from the ovarian stroma, which consists of two layers — the outer or *tunica externa*, and the inner or *theca*. The theca is richly supplied with blood-vessels, and consists of looser connective tissue than the tunica externa. Large numbers of the cells of the inner layers of the stratum granulosum now undergo liquefaction and break down, thus producing a quantity of fluid. Transudation of fluid probably also occurs from the vessels of the theca, and from these two sources the *liquor folliculi* is formed. The cells which immediately surround the ovum do not liquefy, but remain, forming the *cumulus* or *discus proligerus*, which is situated opposite the point where the follicle afterwards ruptures. At the same time a clear, delicately-striated membrane is formed around the ovum as a cell-envelope; this is the *zona pellucida* or *z. striata*, and it is now generally regarded as arising from the follicular cells. Between the zona pellucida and the cell body a microscopic interval, however, remains, called the *perivitelline space*. Before the formation of its cell-envelope the ovum increases considerably in size, and the greater part of its protoplasm becomes coarsely granular, while the nucleus becomes excentric in position; afterwards no further increase in size occurs. It will thus be seen that a fully-formed Graafian follicle consists of the following parts, passing from without inwards:—(1) Tunica externa; (2) Theca; (3) Membrana granulosa; (4) Liquor folliculi; (5) Cumulus proligerus; (6) Ovum.

Ripe Follicles.—In the process of ripening the follicle undergoes further increase in size, bulges upon the surface of the ovary, and finally ruptures at some point upon its exposed surface. Ripe follicles are sometimes met with before puberty, but are most numerous during the menstrual period of life; there is no evidence,

however, to show that any definite relation subsists between the process of menstruation and the ripening of the Graafian follicles. The mechanism of rupture appears to be as follows:—Progressive increase in size of the follicle is due to the activity of the membrana granulosa and to continuous accretion of fluid. The follicle protrudes upon the surface of the ovary, since that is the direction of least resistance. The actual bursting of the follicle depends upon degenerative changes in the follicular epithelium, which weaken the wall so as to render it unable to resist the pressure of its contained fluid. The cells of the cumulus are included in the degenerative change, so that when the follicle bursts the ovum is detached and so escapes. The exact nature of the degenerative process is undetermined, but fatty metamorphosis plays a considerable part in it.

According to the recent observations of Nagel (Fig. 1), the human ovum when discharged from the Graafian follicle is furnished with a well-defined covering of follicular cells arranged in two layers, the outer layer consisting of several rows of small, round, or polygonal, closely aggregated cells, the *zona granulosa*, within which is a double row of columnar cells called *the corona*. This cellular covering protects the ovum during its migration from the ovary to the uterus, and is gradually dispersed in its transit through the oviduct. Stripped of its protective layers the ovum is a simple cell consisting of a cell-envelope, the *zona pellucida*, a cell body, *the yelk or vitellus*, a nucleus, the *germinal vesicle*, and a nucleolus, the *germinal spot*. It measures 2 mm. (about $\frac{1}{5}$ of a line) in diameter. The zona pellucida is a delicate, finely-striated membrane believed to be derived, like the temporary coverings of the ovum, from the follicular cells. Between it and the vitellus is a microscopic space called the *perivitelline space*. The essential portion of the ovum is the central mass of protoplasm with its nucleus, representing the vitellus and germinal vesicle. By the earlier anatomists Graafian follicles were mistaken for ova, and it was not until 1827 that the true ovum cell was discovered by von Baer. At this stage the ovum is unripe and incapable of fertilisation; certain preparatory changes now occur in it, which, in some unknown manner, render it fit for the reception of a spermatozoon.

Ripening of the Ovum.—This process probably occurs during the passage of the ovum through the Fallopian tubes. After being discharged from the Graafian follicle the ovum is carried into the tube by the peritoneal currents set up by the action of the cilia exposed upon the fimbriæ which surround the abdominal ostium; its transit through the tube is also effected by the action of the cilia, lashing from within outwards or towards the uterus. The length of time occupied in this journey in the human species is unknown, but it has been observed to take eight days in bitches, and in some other mammals three to five days (Francke). Hyrtl discovered a ripened ovum in the uterine end of the tube of a girl three days after the cessation of menstruation, but as we do not know that menstruation and ovulation are coincident processes no conclusion can be drawn from such observations.

The ripening process consists in certain

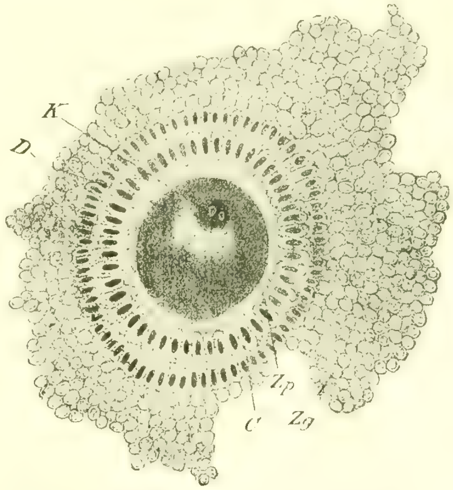


Fig. 1.—*Zg*, zona granulosa; *Zp*, zona pellucida; *C*, corona; *D*, vitellus or yelk; *K*, germinal vesicle. (Playfair.)

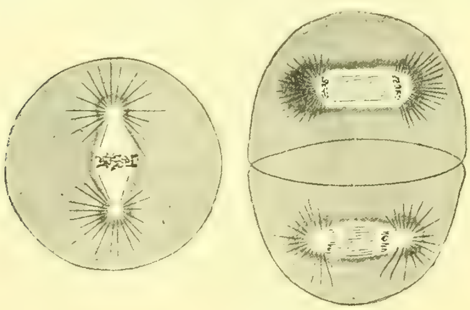
changes in the ovum which result in the formation and extrusion of the *polar bodies* and the appearance of the *female pronucleus*. The process has been minutely studied in the transparent ova of sea-urchins and star-fishes, and among vertebrates in those of the amphioxus. First the germinal vesicle approaches the periphery of the cell and becomes indistinct in outline; mitotic changes then occur in it, while a number of radial striæ appear in the surrounding protoplasm. A minute portion of the germinal vesicle is then detached and extruded into the perivitelline space, along with a ring of protoplasm from the surrounding yelk. A second precisely similar body is also extruded either at the same or at a neighbouring spot. These are the *polar bodies*. In vertebrate animals the second polar body is not separated until after fertilisation. The nucleus now recedes towards the centre, and is henceforth known as the *female pronucleus*. The ovum is now prepared for the coming of the spermatozoa.

Fertilisation of the Ovum.—By "fertilisation" is understood the union of the ovum with the spermatozoon. It is now agreed that this event takes place in the wide outer extremity or infundibulum of the Fallopian tube. To this locality the spermatozoa make their way by their own locomotive powers which are known to be considerable. Experimentally human spermatozoa have been observed by Hyrtl to

travel at the rate of 2 to 7 mm. per minute. In guinea-pigs they have been discovered in the cervix uteri fifteen minutes after copulation. No less important is the fact that their vitality is also great, for they have been found in a living state in the cervix uteri and in the Fallopian tube of woman nine days after copulation (Dührssen). It is probable from these considerations that spermatozoa may lie in wait for the ovum for several days, and that on the appearance of the latter in the Fallopian tube fertilisation immediately occurs. This view receives incidental support from the clinical fact that intercourse is more likely to prove fruitful during the few days preceding the expected onset of menstruation.

In most animals only one spermatozoon is concerned in the fertilisation of a single ovum. In abnormal conditions of the ovum, however, two or more may enter it (*super-fertilisation*). This result can be experimentally produced by slightly damaging the ovum by alterations of temperature, or the action of various drugs (Hertwig). In the mouse, as soon as a single spermatozoon has entered it the ovum throws off a double membrane from the periphery of the yelk which prevents the entrance of others (Kollman). The entrance of the spermatozoon excites great disturbance in the yelk, which contracts and becomes irregular in outline. This disturbance soon passes off, and it is then seen that the tail of the spermatozoon has disappeared, dissolved apparently by the yelk. The remaining portion, or head, is now called the *male pronucleus*, gradually it approaches the female pronucleus, and fusion then occurs between the two, resulting in the formation of a single body, henceforth called the *segmentation nucleus*. The ovum is now fertilised and prepared to enter upon its new career.

Migration of the Fertilised Ovum.—By the action of the cilia of the tubal epithelium, lashing from within outwards, the ovum is



i. ii.
FIG. 2. (Playfair.)

carried into the uterine cavity. After much discussion, it has now been established by Hofmeier and Mandel that the cilia of the uterus move in the same direction as those of the tubes, viz. from within outwards. The older view, that those of the uterine epithelium worked in an opposite direction, thus promoting the advance of the spermatozoa, was therefore an error. The time occupied in reaching the uterus is unknown, nor do we know to what stage the segmentation process next to be described has advanced when the fertilised ovum becomes engrafted upon the uterine decidua. No observations have ever been made in the human species upon the process of fertilisation and segmentation, or the formation of the germinal layers; the following description is based upon observations made on lower animals.

Segmentation of the Ovum.—After the fusion of the male and female pronuclei a short pause occurs, and the process of *segmentation* then commences. The segmentation nucleus first shows karyokinetic changes, and then divides into two halves, which retreat towards opposite poles of the ovum, while radial lines appear in the protoplasm around them (Fig. 2, i.). An equatorial line of division is next formed in a plane between the two nuclei, which divides the whole ovum into two cells. The same changes now occur in each of the new cells (Fig. 2, ii.), and thus by a process of binary division the ovum multiplies into 2, 4, 8, 16, 32, etc. cells. In the mammalia the whole ovum is involved in the segmentation process, and is therefore called *holoblastic*, in contradistinction to that of fishes, reptiles, and birds, in which only a portion is involved, the ovum being called *meroblastic*. In this manner, then, the mammalian ovum is converted into a solid cluster or globe of cells often called the *morula* or *muriform body* (Fig. 3, *a*). In the rabbit (van Beneden) the cells of the muriform body are of two kinds, clear peripheral cells and granular central cells (Fig. 3, *b*). A crescentic cleft soon forms between the peripheral and central cells, which becomes filled with fluid, and by rapid increase in this fluid the morula increases enormously in size and becomes converted into a globular sac, called the *blastodermic vesicle*. The original peripheral cells of the morula have been flattened by pressure from within, and form a single layer within the zona pellucida; the central granular cells are found in contact with the outer cells at one pole of the vesicle, but they do not form a continuous layer (Fig. 3, *c*).

The next stage is the formation of the *blastoderm*. The deep granular cells proliferate and spread out within the outer layer, forming a patch at first circular and afterwards oval in shape; the two layers together form the *bilaminar blastoderm*, and the oval area of the surface of the blastodermic vesicle where they are found is called the *embryonic area* (Fig. 3, *d*). Next a delicate longitudinal ridge, formed by proliferation of the cells of the outer layer (*ectoderm*), may be traced along the embryonic area; this is the *primitive streak*, and upon it appears a shallow, longitudinal groove, the *primitive groove*. Along the line

of the primitive streak the cells of the inner layer (*entoderm*) also proliferate and come into contact with the ectoderm, a narrow cleft separating the two layers elsewhere. From this line of contact a third layer of cells is developed, which spreads out in the interval between the outer and inner layers; it is derived mainly from the ectoderm, but also in part from the entoderm. The *blastoderm* is now *trilaminar*, the layers being termed *epiblast*, *mesoblast*, and *hypoblast* from without inwards; from these layers all the tissues of the body are ultimately developed.

Soon after the appearance of the mesoblast a shallow, longitudinal groove, the *medullary groove*, is formed on the embryonic area in front of the primitive streak. It is produced by the formation of two parallel folds of epiblast called the *medullary folds*. These grow over to meet one another, and coalesce in the middle line, forming an included canal with an epiblast lining, the *neural canal*, from which the central nervous system is developed (Fig. 4). During these changes the primitive streak disappears. While the neural canal is developing there is formed beneath it a solid rod of hypoblast cells, the *notochord* (Fig. 4). At the sides of the neural canal a great development of mesoblast cells takes place, forming large lateral masses which become marked out by transverse grooves into a row of solid blocks called the *mesoblastic somites*. From these cell-masses most of the skeletal and muscular tissues of the body are developed. At the same time that the mesoblastic somites are formed the outlying parts of the mesoblast split into two layers: the outer becomes applied to the epiblast, and the two together form the *somato-pleure*; the inner becomes similarly applied to the hypoblast, the two together forming the *splanchno-pleure*. The space between the somato-pleure and splanchno-pleure is the *cœlom* or primitive body-cavity (Fig. 4).

It will be understood that the abovementioned changes have taken place upon a certain small area of the blastodermic vesicle, and of course within the zona radiata. The next step is the delimitation of the outlines of the embryo by the formation of the anterior, posterior, and two lateral limiting sulci. At the same time the embryonic area begins to recede towards the centre of the blastodermic vesicle—sinking that is into its interior, and leaving a space between it and the enclosing zona radiata. A period has now been reached at which certain structures designed to provide for the protection and nutrition of the embryo make their appearance. These are the fœtal envelopes or membranes called the *chorion* and the *amnion*. The function of the amnion is protective; the chorion serves to bring the embryo into union with the maternal structure, its function being therefore mainly nutritive.

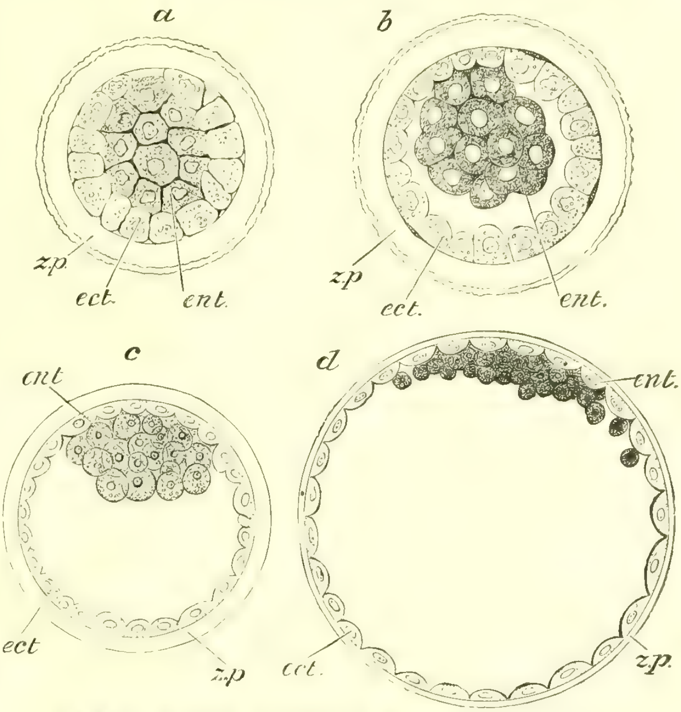


FIG. 3.—*ect.*, Ectoderm; *ent.*, entoderm; *zp.*, zona pellucida. (Playfair.)

The fœtal membranes are developed from folds of the somato-pleure which grow up from the head and tail ends and lateral boundaries of the embryo, and pass over its dorsal surface in the space between it and the zona radiata. Gradually they coalesce, forming a closed hood, which consists of a central core of mesoblast with an internal and external covering of epiblast (Fig. 4). The mesoblast layer now splits into two, and fluid collects between the two halves; there is thus formed a double hood, the outer layer consisting of an outer covering of epiblast with mesoblast within, and the inner layer consisting of an inner covering of epiblast with mesoblast outside it. The outer layer becomes the *chorion*, the inner layer the *amnion*. The chorion now becomes closely applied to the zona radiata, and fuses with the extra-embryonic portion of the epiblast, *i.e.* that portion of the wall of the blastodermic vesicle which was not concerned in the formation of the embryonic area. The amnion closely invests the dorsal surface of the embryo, and a small quantity of fluid collects within it, but during the early weeks of development the amniotic sac is very

small; the fluid it contains is the *liquor amnii.*

The body-folds (somato- and splanchnopleure) now grow over towards the ventral surface, thus pinching off the embryo from the remainder of the blastodermic vesicle. The effect of this change is to carry the line of origin of the amniotic folds, at first dorsal in position, farther and farther over to the ventral surface of the embryo; and as the cœlom becomes closed by union of the body-folds in the middle line, the amnion comes to be continuous with the body of the embryo at one point only, which is the last to close, viz. the umbilicus.

It will be seen that the effect of the development of the amnion and chorion is to isolate the embryo in the interior of the blastodermic vesicle. Direct communication with the periphery is, however, maintained by the development of the structure known as the umbilical cord. Important observations have been made upon the development of this structure in the human species by His. Among the lower mammals the *allantois* plays an important part in the development of the cord, and to His belongs the credit of first pointing out the differences to be found in man. In mammals generally the allantois grows out from the hinder end of the primitive gut, forming a bladder-like structure of considerable size outside the body, known as the *allantoic vesicle.* Over this structure vessels pass to the chorion. In man, however, the allantois is much less important, forming a narrow canal which never grows out far enough to reach the chorion, and does not form an expanded vesicle outside the body. It lies in a band of somato-pleure tissue which passes from the ventral aspect of the caudal extremity of the embryo to the wall of the vesicle where it is continuous with the chorion. This structure His named the *ventral stalk.* It carries vessels from the posterior end of the primitive aorta to the chorion, and these vessels may be detected at a period before the allantois has been formed. At first springing from the caudal extremity, its point of origin gradually approaches the centre of the body, the change in relation being due to the development of the pelvis and lower limbs behind it. The umbilical vesicle, which in man is small and unimportant, with its omphalo-mesenteric duct, also fuses with the ventral stalk, and thus becomes one of the constituent structures of the cord. Finally, as the line of origin of the amnion is carried over to the ventral aspect of the embryo, the cord receives a covering from this membrane also. By some observers, however, the epithelial covering of the cord is regarded as modified fœtal skin (Minot). The portion of the allantois within the abdominal cavity forms the bladder and urachus.

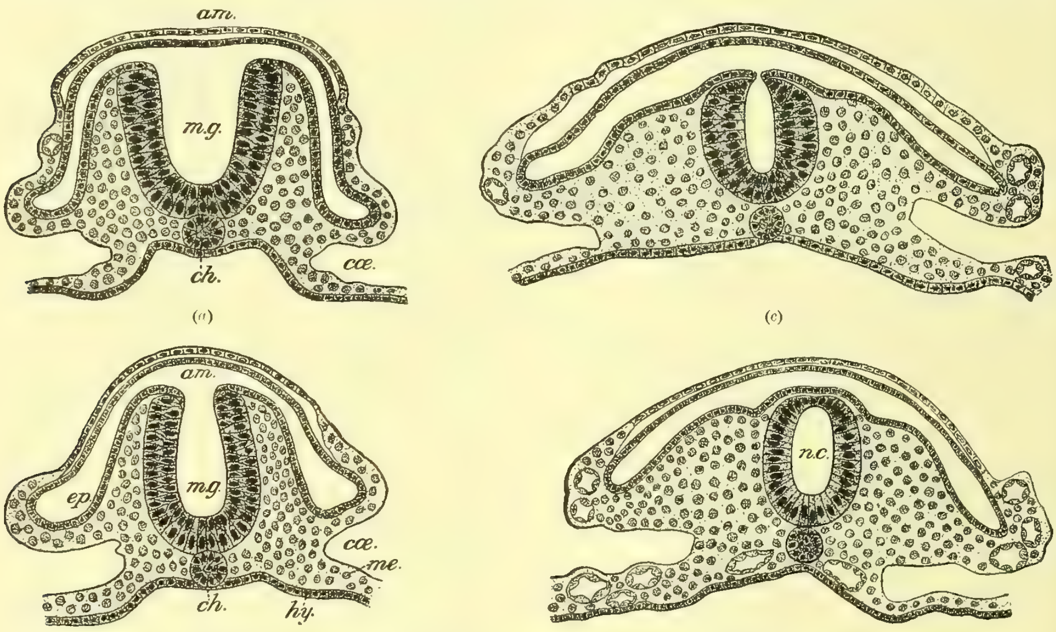


FIG. 4.—*am.*, Amnion; *mg.*, medullary groove; *ch.*, notochord; *cœ.*, cœlom; *ep.*, epiblast; *me.*, mesoblast; *hy.*, hypoblast; *nc.*, neural canal. (Playfair.)

Up to this period we have been obliged to rely entirely upon facts observed in the development of lower animals. There is good reason to believe, however, that the stage at which we have now arrived is reached in the human embryo at the end of the first week of development. This statement is based upon recent observations by Leopold, which will be referred to more fully later on. The embryo now lies

enclosed entirely within its bag of membranes, and is connected with the outer one (chorion) by a vascular stalk. The zona pellucida has disappeared, leaving the chorion exposed upon the wall of the ovum, and the latter has thrown out numerous branching processes containing vascular twigs from the allantoic vessels; these processes are the chorionic villi, and at the end of the first week they are found upon almost the whole surface of the chorion. The next important step is the formation of attachments to the maternal structures, by which the developing ovum is retained in position and preserved from disruption. These attachments are provided by the development of the decidua and the union of the chorionic villi with this membrane.

The Decidua.—The time-honoured view that menstruation in some way prepares the uterus for the reception of a fertilised ovum has much in its favour; but that its influence is not essential is proved by the fact that pregnancy not infrequently occurs quite independently of it. The structure of the endometrium in the quiescent stage and during the menstrual process has been described elsewhere. The decidua differs from the endometrium in the following points:—1st, The connective tissue corpuscles become converted into large cells of epitheloid type with one or two globular nuclei; these are called the "*decidual cells*," and are rarely if ever found in the endometrium except in connection with pregnancy. 2nd, The glands proliferate and become dilated, especially in their deeper parts. 3rd, In consequence, the decidua becomes roughly divided into two layers, a superficial *compact* layer and a deep *ampullary* layer. 4th, The vascularity of the membrane is greatly increased, and numerous small interstitial hæmorrhages are formed.

It is usual to distinguish three divisions of the decidua; the *decidua serotina* is the portion which corresponds to the site of attachment of the ovum and partakes in the formation of the placenta; the *decidua reflexa* is the portion which at a very early period grows over the ovum so as to enclose it; the *decidua vera* is the remaining portion, covering the greater part of the uterine surface, which at first has no direct relation to the ovum at all. All parts of the decidua are essentially alike in structure as well as in origin. No decidua is formed in the cervix.

Leopold has described with great care a seven days' human ovum which he discovered by accident in a uterus removed for cancer of the cervix (Fig. 5). He found the uterine mucosa much thickened in the body, but unaltered in the cervix. The ovum was completely enclosed in a fold of the membrane, so that we know that even at this early period the reflexa is fully formed. Folds of the decidua grow up around the ovum and meet over its free pole so as to completely invest it. From Leopold's illustration (Fig. 6) it is apparent that the decidua and the chorion are separated by a considerable space, except at the two poles of the ovum; at the pole of attachment a serotinal process is in direct contact with the chorion, and at the free pole, where there are no villi, chorion and reflexa are united over a considerable area. The space thus resulting (*chorio-decidual space*)

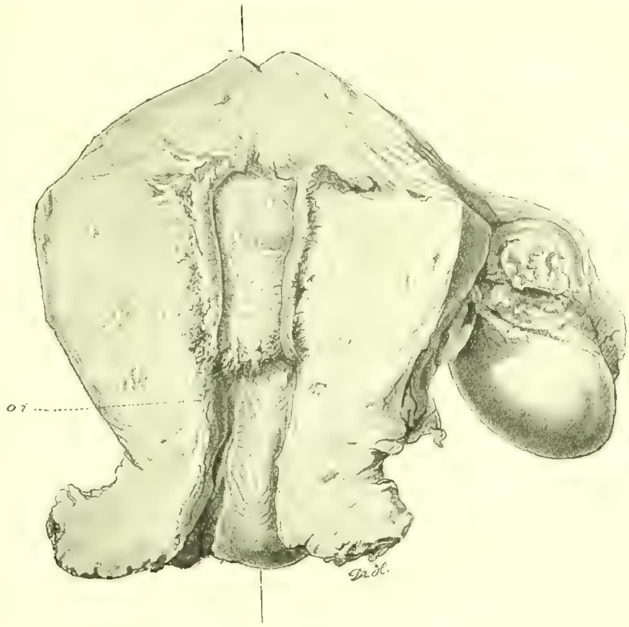


Fig. 5.—*oi*, Os internum. (Playfair.)

is occupied by the chorionic villi, which are seen in longitudinal, transverse, or oblique section in the drawing. Most of the villi are free; some are attached to the decidua by their tips (Fig. 6, *h*); others have penetrated it deeply, and are seen in cross-section embedded in decidual tissue (Fig. 6, *f*). Around the villi are masses of red and white blood corpuscles, and one or two delicate capillaries are seen opening through the serotina into the chorio-decidual space (Fig. 6, *e*). At a somewhat later period (end of second week), as Leopold has shown in a second case, the whole chorionic surface is beset with villi, and the chorio-decidual space is continuous around the entire ovum.

These points are of great interest and importance. They indicate that at this early period chorionic villi are attached to the highly vascular decidual membrane which entirely surrounds the ovum, while maternal blood is poured into the chorio-decidual space, thus bringing the fœtal tissues into direct contact with the maternal blood. A simple form of placentation is thus established, which by the end of the

second week involves the whole superficies of the ovum, thus corresponding to the diffused placenta of the sow, the mare, the cetacea, etc. In Leopold's first case traces of vascularisation were found in some, but not all of the villi, and it is therefore probable that the fœtal circulation is at this stage incomplete, and transference of nutriment by osmosis from the maternal to the fœtal blood current cannot occur; but doubtless the villi are able to absorb directly from the effused maternal blood without the aid of an active circulation. By this means time is gained for the development of the highly specialised discoidal placenta, which is well outlined by about the sixth week.

Development of the Placenta.—We do not know the precise period at which villi make their appearance on the human chorion, or the precise period at which these structures first come into relation with the maternal tissues. There is reason to believe that they first appear on the pole of attachment of the ovum, and here the union of fœtal and maternal structures may follow very quickly upon implantation. When formed they are probably at once vascularised through the allantoic vessels. By the end of the second week they are well-formed branching structures, which cover the entire chorion. The part of the chorion which forms the placenta corresponds to the pole of attachment of the ovum, and is often called the *chorion frondosum*; the extra-placental portion is called the *chorion lœve.* The latter is so-called because it was once thought to be non-villous; later on, it is true, it becomes converted into a smooth membrane by the disappearance of its villi, but at first the villi are fairly equally distributed over the whole chorion. The formation of placenta simply consists in the specialisation of a part of the chorion to do the work which in the earlier stages is done by the whole. On the chorion frondosum the villi increase greatly in size, in number, and in the complexity of their branchings, while the extra-placental villi gradually disappear; at the same time important changes occur in the underlying serotina. The placenta, therefore, consists of fœtal and maternal elements which must be considered separately.

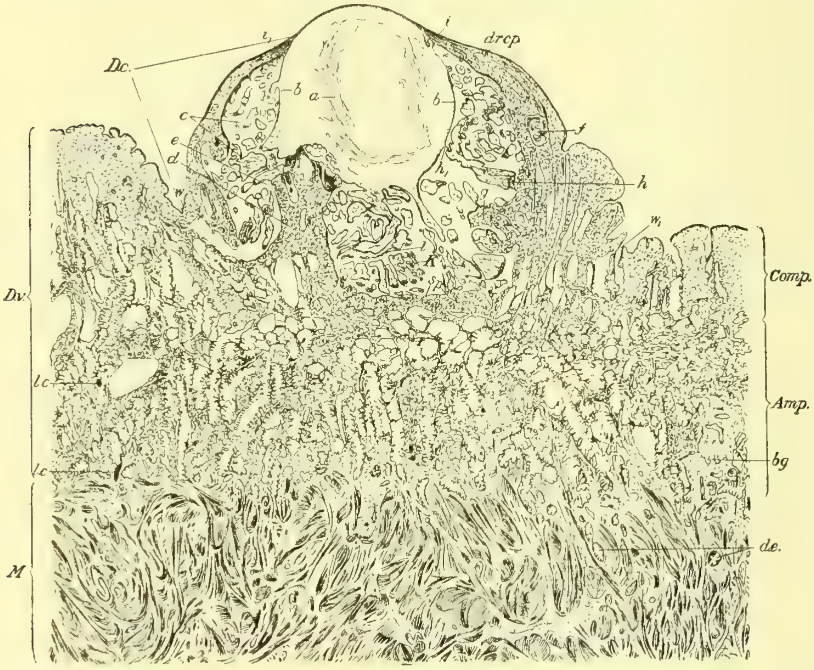


Fig. 6.—*Dv.*, Decidua vera; *M*, muscularis; *Comp.*, compact layer; *Amp.*, ampullary layer; *Dc.*, decidua reflexa; *a*, amnion; *b*, chorionic sac; *c*, chorionic villi; *d*, decidua serotina; *e*, mouth of maternal vessel; *h*, attached villi; *f*, embedded villi. (Playfair.)

The Fœtal Elements of the Placenta.—By the end of the second month the chorion frondosum consists of a membrane underlying the amnion with a dense forest of tree-like structures growing from its outer surface. These are the chorionic villi (Fig. 7). It is probable that from their first appearance the villi are branched; in the placenta they are much divided, their ramifications being quite irregular in size, shape, and direction. Consequent upon the latter point considerable interlacement of neighbouring villi may be found . The final divisions (terminal villi) are more or less club-shaped structures with a slightly constricted base of attachment. The villi occupy the chorio-decidual space, but there are wide intervals between them, forming the system of intervillous spaces through which the maternal blood flows. Many of the villi are attached to the serotina by their tips, some penetrate it for a considerable distance, but the bulk of them are free. All portions of the placental chorion consist of the following structures: (1) an epithelial covering; (2) a connective tissue stroma; (3) a system of blood-vessels.

(1) *The chorionic epithelium* clothes the outer (uterine) surface of the chorionic membrane and villi. It consists of two distinct layers: an *outer layer* of multinucleated protoplasm, or plasmodium, in which no cell outlines can be distinguished, and which is not therefore truly

a cellular layer, and an *inner layer* of large, well-defined cells, with large oval nuclei. The former is called the *plasmodial* layer; it has received many other designations, of which *trophoblast* (Clarence Webster), and *syncytium* (German authors) are the most important. The latter is called the *cellular layer*, or the *layer of Langhans.* It is probable that both these layers arise in the fœtal epiblast, although many observers regard the plasmodial layer as maternal in origin, while others regard the cellular layer as derived from mesoblast. This histogenetic problem cannot, however, be discussed in this article.

During the early months of gestation the plasmodial layer shows great activity. From it spring numerous buds, elongated or club-shaped, conical or rounded, and having the same structure as the layer from which they arise. The plasmodial buds represent the first stage in the process of multiplication of the villi. Their protoplasm soon becomes freely vacuolated, and the stroma then grows into them, carrying with it vascular twigs from the allantoic vessels, thus completing the structure of the new villi.

(2) *The stroma* is a delicate reticulum of connective tissue supporting the blood-vessels. In the larger chorionic divisions it is more compact, resembling loose fibrous tissue, and is especially well marked around the larger arteries. The interstices probably represent lymphatic spaces.

(3) *The blood-vessels* are the terminal ramifications of the allantoic or umbilical arteries and veins; they pass into every division of the chorion, and in an injected specimen a delicate thread may be often traced passing into a plasmodial bud which has just become vascularised. In the larger divisions the vessels lie in the axis; arteries and veins run side by side, the latter being distinguished by their thinner walls and larger calibre. In the terminal villi capillaries alone are found; they are placed for the most part immediately beneath the epithelium, where they run a tortuous course and anastomose freely. They form wide channels in which five or six red corpuscles can lie abreast, and they occupy such a large extent of the stroma that the villus appears to be as full of blood as a soaked sponge. The capillary walls are formed of a single layer of delicate endothelium, and nothing intervenes between them and the maternal blood except the chorionic epithelium.

The Maternal Elements of the Placenta.—The part played by the serotina in the development of the placenta is an important one. In the first place, it is the medium through which the maternal circulation, through the intervillous spaces, becomes established; and in the second place, it serves to attach the fœtal elements firmly to the uterine wall.

The chorionic villi are the principal agents in the production of the changes which result in the establishment of the intervillous circulation. It will be remembered that in the seven days' ovum described by Leopold some of the villi had penetrated the decidua. During

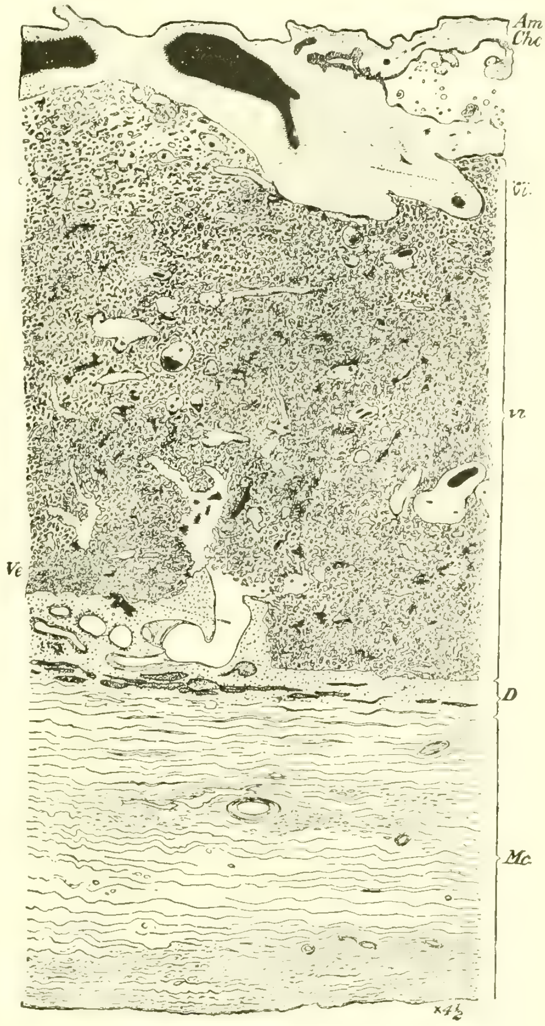


Fig. 7.—Section through a normal seven months' placenta, *in situ*. (After Minot.) *Am.*, amnion; *Cho.*, chorion; *Vi.*, primary villus, vessels injected; *vi.*, smaller villi; *Ve.*, maternal vein opening into intervillous spaces; *D*, decidua serotina; *Mc.*, muscular wall of uterus. (Playfair.)

the second month this process goes on extensively in the serotina; the villi do not enter the glands, as was once supposed, but penetrate the tissues by literally eating their way through. Around the invading buds the decidual cells necrose and become absorbed, the fœtal epiblast appearing to exert some potent influence upon them. The plasmodial buds act as pioneers, and numbers of them may be seen embedded in the decidua in advance of the villi. In its new position each bud is vascularised in the manner already indicated, and the new villus in its turn

throws off new buds. These changes occur most markedly in the compact superficial layer of the serotina, but occasionally buds may be found to have bored their way right through the decidua into the uterine musculature.

While the invasion of the serotina by the villi is proceeding, large hæmorrhages are formed in the membrane. Many capillaries, or even larger vessels, are penetrated by the plasmodial buds, and from these extravasation occurs, leading to the collection of large blood-lacunæ in which buds and villi may be found lying. These lacunæ finally rupture into the chorio-decidual space, and thus throw fresh maternal vessels into communication with it. The larger lacunæ have often been shown to have an artery, and one or two veins communicating with them. The precise period at which the maternal circulation through the chorio-decidual space is established is unknown, nor are the precise steps in the further process of development finally determined. It is doubtful if there is any true maternal circulation in the temporary diffused placenta; there is a good deal of clotting in the chorio-decidual space in very young ova, and the villi probably absorb nutriment from the effused blood. In the fully-formed placenta it is, however, easy to show that large maternal arteries and veins open directly upon the serotinal surface. To Waldeyer belongs the credit of first demonstrating this fact by injecting the uterine arteries and veins in the cadaver of a pregnant woman. The arteries preserve their characteristic coiling course through the serotina (Fig. 7, *a*), and then open somewhat abruptly into the spaces; the veins run an oblique course and open by long oblique mouths. Arterial may be distinguished from venous openings by (1) the thickness of the walls; (2) the direction of the opening; (3) the fact that villi are washed away from the mouths of the arteries by the blood current, while they may be drawn by the same force into the mouths of the veins.

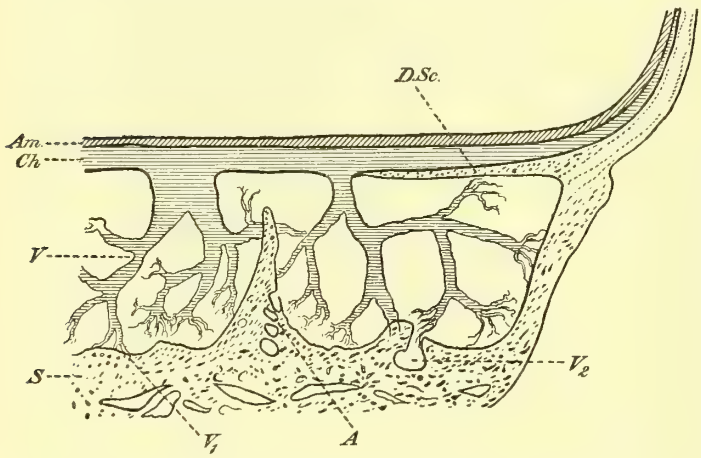


FIG. 8.—*Am.*, amnion; *Ch.*, chorion; *V*, villi; *S*, decidua serotina; V_1, attached villi; V_2, vein; *A*, artery; *D.Sc.*, subchorionic decidua. (Playfair.)

While these changes are progressing the ovum is held to the uterine wall chiefly by the reflexa; later on the placenta becomes firmly united to the uterus and constitutes its main attachment. The delicate character of the early attachment of the ovum is an important factor in determining the frequency with which abortion occurs at this period; when the placenta is fully formed it is much less easily disturbed. The fœtal and maternal elements of the placenta are united by the following means (Fig. 8):—(1) Many of the villi remain embedded in the serotina; (2) Processes grow up from the serotina among the villi, sometimes reaching as far as the chorionic membrane, and many villi are united to them by their tips; (3) From the serotinal margin a process of decidual tissue grows towards the centre of the placenta in contact with the outer surface of the chorionic membrane; this layer is called the *subchorionic decidua*, and may be traced in the ripe placenta for one or two inches (Fig. 8, *D.Sc.*) Being continuous around the whole circumference of the placenta it serves to strengthen the attachments where they are weakest, and also to limit circumferentially the general system of intervillous spaces through which the maternal blood flows.

The circulation through the intervillous spaces is probably not rapid. The coiling course of the uterine arteries diminishes the force of the blood stream, and therefore the *vis a tergo* in the spaces; the outflow is largely promoted by the intermittent uterine contractions, which have the effect of aspirating their contents into the veins. A slow current is an advantage in allowing time for osmotic interchanges.

The placenta grows very rapidly during the first few weeks after its formation; it encroaches more and more over the uterus until by the end of the third month it occupies one-fourth of the total uterine surface. At the same time the ovum becomes large enough to entirely fill the uterine cavity; the decidua reflexa comes into apposition with the vera and fuses with it, forming a single membrane. The ovum is thus directly supported by the uterine walls, and from this point onwards uterus and ovum grow *pari passu*, and the proportion of the uterine surface occupied by the placenta remains at one-fourth. The placenta is limited at its margin by the line of reflection of the decidua (reflexa) upon the ovum. Previous to

the end of the third month this line of reflection moves with the growth of the placenta, passing farther and farther outwards to enclose a gradually increasing area. During this period, therefore, the reflex grows with the ovum; afterwards it is unnecessary, and becomes thinned and atrophied from pressure. The placenta, however, continues to increase in size proportionately with the uterus, while in thickness it undergoes progressive increase within the area mapped out for it.

The atrophy of the extra-placental chorionic villi, which is complete by the end of the third month, is chiefly due to the withdrawal of their blood-supply consequent upon the development of the placenta. Somewhat later, when the ovum fills the uterus, the extra-placental chorio-decidual space is obliterated by pressure, and the atrophied villi become surrounded by rings of fibrin deposited by clotting from the maternal blood. In this form they may always be found in the membranes of a young ovum, where they appear to be embedded in atrophied decidual tissue. Near the placental margin traces of them may be found at term.

The Placenta at Term.—When shed from its uterine attachments the placenta is a flat, circular, or oval mass measuring from six to eight inches in diameter, three-quarters to one inch in depth at the thickest part, which corresponds to the insertion of the cord, and weighing about 16 ounces. The margin is thinner and firmer than the centre, and passes somewhat abruptly into the chorion læve.

The fœtal surface contrasts strongly with the maternal. It is covered with a smooth, shining membrane, the amnion, which can be readily stripped off up to the insertion of the cord. Beneath it lie the surface branches of the umbilical vessels. The arteries divide at once upon reaching the placenta, and continue to branch irregularly as they approach the margin, but the terminal divisions never quite reach the edge. The veins accompany and often cross the arterial branches. The latter plunge vertically into the placental tissue, then run for a short distance horizontally, and then dip downwards again, thus forming a series of steps in their course. Thus they pass into the ramifications of the villi in company with the veins. The surface of the chorion exposed after removal of the amnion is smooth and polished; on incising it a membrane of the thickness of about one line is seen to form the surface, and from its deep aspect springs the mass of spongy tissue representing the chorionic villi. Attached to the deep surface of the membrane are large numbers of small greyish or yellowish nodules, seldom larger than half a pea. These are masses of fibrin the origin of which will be referred to under "Pregnancy, Diseases of Placenta."

The uterine surface is dark red in colour, dull in appearance, and divided by sulci into a variable number of more or less quadrangular areas called *cotyledons.* On close inspection a thin, greyish, mottled membrane may be observed upon the surface; in parts it is incomplete, leaving the deep red, spongy tissue exposed. It usually feels rough and gritty, and often little calcareous plates may be seen with the naked eye. It represents the shed portion of the serotina; it passes into all the inter-cotyledonary sulci, and is continuous with the decidua at the placental margin. It cannot be stripped off like the amnion. If the placenta be floated under water the torn ends of the coiling serotinal arteries may sometimes be seen. Around the margin runs a large venous channel, the *circular sinus;* this returns a part of the blood from the intervillous spaces; it is usually interrupted in several places.

When incised the cut surface of the placenta is seen to be of a dark mottled purplish colour, and traversed by numerous thin, greyish bands representing the larger chorionic branches. A great deal of blood slowly exudes from an incision into the placenta, which is mostly maternal blood from the intervillous spaces. If a stream of water be turned upon the cut surface the spaces are washed out, and the arborescent villi then become evident, appearing as delicate, greyish branching threads.

Degeneration in the Placenta at Term.—To understand the structure of the ripe placenta, we must remember that we are dealing with an organ which is intended to serve only a temporary purpose. It is, in short, a caducous organ, and when its purpose is fulfilled it is shed like a withered leaf. Now all caducous organs degenerate before they are cast off, the immediate cause of their shedding being that they are degenerated. So the ripe placenta is a senile structure no longer fitted to play its physiological part, and is therefore repudiated by the organism. This failure of vitality commences long before the approach of the term of gestation, and results in marked structural alterations in the placenta. The importance of a right estimation of these changes is of great importance, for it is obviously impossible to make any progress with the study of its diseases until the normal structure of the organ has been fully worked out. It is convenient to consider separately the changes in the fœtal and maternal elements.

Changes in the Fœtal Structures.—The initial change affects the allantoic (umbilical) vessels; all others are secondary to, and dependent upon, this one. Stated briefly, they result in the obliteration of extensive arterial tracts, and consequent arrest of the circulation in large tracts of villi. The terminal and medium-sized arterioles are first affected; the largest arteries suffer later, while the veins and capillaries remain unaffected until the circulation through

them is entirely suspended by destruction of the feeding arterioles. The change chiefly affects the intima (endarteritis) with some participation of the adventitia (periarteritis). Arteries showing these changes may be found in every ripe placenta; they are most numerous in the marginal cotyledons, and become progressively fewer towards the centre, but the total number of branches affected is relatively small. It is a little difficult to determine the precise period at which these changes begin, but it is probable that they may be found as early as the beginning of the seventh month, and appear to be slowly progressive during the three last months of gestation.

From these changes results a progressive diminution in the blood-supply of the villi fed by the affected arterioles. The first structure which suffers therefrom is the epithelial covering. Many villi show in places complete atrophy of the plasmodial layer, the nuclei having disappeared, and nothing remaining but a thin structureless band. This change only occurs in patches, and intermediately the layer may be fairly normal in appearance. The layer of Langhans has entirely disappeared at this period, but it is doubtful whether its disappearance is due to the arterial changes, it probably precedes them. In addition to these atrophic changes the epithelium also undergoes a peculiar form of degeneration which has been the subject of much dispute, and is generally known as "fibrinous degeneration." It affects parts in which atrophy is not marked. An opaque fibrillated material, which stains much more deeply with ordinary reagents than the unaffected parts, takes the place of the plasmodial protoplasm. It possesses many of the staining reactions of fibrin, but also many affinities with hyaline material, and probably results from a process allied to coagulation-necrosis. Upon the degenerated areas fibrin is now deposited from the maternal blood in the spaces, and often considerable heaps of this material are found. Owing to the similarity of their staining reactions it is difficult to say where the fibrin ends and the degenerated epithelium begins; the fibrin is, however, usually reticulated, which the degenerated epithelium is not. In the ripe placenta these changes may be found upon any of the surfaces exposed to the maternal blood, but the proportion of villi affected is small, the greater number remaining free. Heaps of fibrin deposited upon neighbouring villi may meet, forming a bridge between them, and filling up the intermediate space. In this manner single villi or clusters of villi may be found completely embedded in aggregations of fibrin, thus forming minute areas of consolidation in the spongy placental substance. It is obvious that villi thus embedded must be functionless, being out of contact with the maternal blood. In point of fact, very large numbers of such functionless villi exist in every placenta at term. For the most part they occur in areas of microscopic size, but at times these areas are large enough to be visible to the naked eye, and have received the name of *white infarcts* from their resemblance to old infarctions of the kidney, spleen, etc. A placental infarct is therefore a consolidated area of functionless placental tissue.

Placental infarcts occur as firm, yellowish white, well-defined areas which stand forth in strong contrast to the deep red, spongy placental tissue which surrounds them. They vary much in size, but are rarely larger than a filbert; at times an entire cotyledon is involved. At the margin they pass abruptly into the placental tissue, but are never encapsuled. They are most numerous on the uterine surface and in the marginal cotyledons; at times they lie buried in the placental substance. The small nodules found on the fœtal surface of the placenta are small infarcts or masses of deposited fibrin. Large infarcts may soften and break down in the centre, and occasionally become the seat of calcareous deposit.

On microscopic examination a relatively large part of the infarct is seen to be made up of fibrin. The villi involved in it have lost their epithelial covering, but often contain capillaries full of stagnant blood. In the larger divisions the final stages of obliterative endarteritis may be observed. The veins and capillaries remain full of blood from failure of the *vis a tergo*; sometimes also small areas of maternal blood are found between the villi. The affected villi rapidly degenerate; the stroma loses its nuclei, and becomes converted into a structureless material which stains very feebly with all reagents. Their contour, however, remains distinct, as they are mostly limited by rings of fibrin. Fatty degeneration is marked both in the atrophied villi and the masses of fibrin. Finally, necrosis affects all structures, converting them into granular debris. Infarcts increase in size by peripheral extension, hence three distinct zones may be distinguished in the larger ones: a *central zone* of degenerated structureless material; a *middle zone* consisting of fibrin and atrophied villi; and an *outer zone* showing the earlier stages of degeneration of chorionic epithelium and deposition of fibrin in the intervillous spaces.

The existence of placental infarcts has been known for a very long time, but their significance has been generally misunderstood. The older writers all regarded them as the result of disease, and a great variety of different morbid processes have been described as causing them. Robin is alone among the writers of forty to fifty years ago in attributing them to the operation of natural causes; he compares the process with that by which the extra-placental villi are disposed of in the earlier months of gestation. As a matter of fact, atrophied villi embedded in

fibrin may be found in the chorion immediately outside the placental margin at all periods of gestation up to term, and the changes seem to be identical with those occurring in infarcts. In the placenta the process is modified by the presence of the maternal blood-spaces.

Changes in the Maternal Structures.—Friedländer first drew attention to the fact that extensive thrombosis occurs in the subplacental uterine sinuses during the later months of pregnancy; his observations have recently received complete confirmation by other observers. As early as the seventh month large thrombi form in these sinuses, commencing in the muscle and gradually extending towards the placenta. The number of sinuses affected is relatively small, and in view of the free anastomoses which exist, it is doubtful if they form any serious obstruction to the outflow from the intervillous spaces. Friedländer believed that the thrombosis was started by large cells of decidual origin which made their way into the veins by diapedesis. The presence of "giant cells" in the affected vessels has been frequently observed, and it is feasible to suppose that the clotting commences around them. It has already been mentioned that epiblast buds of the chorionic villi have the power of penetrating the decidual vessels, and it may be that some of them are carried by the blood stream into the muscular layer and effect a lodgment there. The precise nature of the giant cells must be regarded as undetermined.

The serotina at term consists of two well-differentiated layers—a superficial compact, and a deep cavernous or ampullary layer. When the placenta is shed separation occurs through the latter, part of the layer going with the placenta, and part remaining attached to the uterus. The wide irregular spaces contained in the ampullary layer represent dilated glands, and even at term retain traces of their columnar epithelium. From the epithelium thus left behind at the placental site and elsewhere the endometrium is regenerated during the puerperium. The compact layer has undergone marked changes. Its superficial strata show that the decidual cells have degenerated, their nuclei have finally disappeared, the outlines of the cells themselves are blurred or lost, and a mass of fibrillated material, easily recognised as fibrin, is deposited upon them from the blood in the maternal spaces. The degenerated decidual cells have much the same staining reactions as those of the degenerated chorionic epithelium, and the two processes are, no doubt, closely allied. This change in the superficial layers of the serotina may be found at any time from the seventh month of gestation onwards to term.

The changes above described in the chorion and decidua, it must be clearly understood, are not pathological. In an organ with a short life-cycle like the placenta, the stages of growth, maturity, and decay succeed one another rapidly. Degeneration of vessels and atrophy of epithelial structures are associated with old age in all organs of the body, and such changes in the placenta are no more pathological phenomena than are the thickened arteries and wasted skin of aged persons. Their effect upon the functional capacity of the organ is difficult to estimate. Their gradual onset and slow progress make them much less serious disturbances than if they ran a rapid course. Probably sufficient placental tissue always remains active to provide adequately for the nutrition of the fœtus. It may be, however, that the onset of labour coincides generally with a period at which the placenta becomes unfit for the further performance in its functions, and in this way placental degeneration may be the most important factor in determining the termination of the period of gestation.

Nutrition of the Fœtus in Utero.—We have seen that from the end of the first week of development onwards, the human ovum receives nourishment from the maternal organism through a placental arrangement, at first general and simple, later, partial and specialised. In what manner the ovum is nourished during the first week, when it increases about twenty times in size, we do not know. In birds sufficient nutriment is stored up in the umbilical vesicle (yelk) to carry the embryo through. In the minute human ovum such cannot be the case. Some recent observations by Hubrecht may throw light upon the question. He has studied with great care the early stages of development of the hedgehog, an animal belonging to a family (*insectivora*) which, with the apes and man, is distinguished among mammals by possessing a discoidal placenta. Hubrecht describes the formation, in the very early stages, of a zone of vascular tissue around the ovum, composed partly of the ectoderm of the blastodermic vesicle, and partly of the adjacent cellular tissue of the decidua. This layer, which he calls the *trophosphere*, is richly supplied with maternal blood. When the vitelline circulation is established primitive mesoblastic villi from the wall of the umbilical vesicle push their way into the trophosphere, and as they contain branches of the vitelline vessels, the effect is to bring the fœtal and maternal blood into close relations with one another. There is, in fact, in the hedgehog a temporary vitelline placenta preceding the formation of the permanent or allantoic placenta. We do not know whether there is any homologue of the vitelline placenta in man, but Hubrecht's observations are highly suggestive.

The fœtal processes of nutrition and oxygenation are carried on through the placenta. As we shall see in a later paragraph, the excretory organs of the fœtus become active at a surpris-

ingly early date, and it is unlikely that the placenta performs any important excretory functions, with the exception of carrying off the carbonic acid of the venous blood which is brought to it by the umbilical arteries. The placental scheme presents an arrangement admirably adapted for osmotic interchanges between the fœtal and the maternal blood currents, the chorionic epithelium representing the dialyser. It is probable that the placenta is merely the vehicle of these interchanges, and not in any sense a glandular organ. Some years ago the theory was much in vogue that the serotinal glands secreted a nutrient fluid termed "uterine milk," which was absorbed by the villi, and thus furnished the fœtus with nutriment. This theory has, however, never been adequately supported by facts. A still older theory that there was a direct interchange of blood by anastomosis between the fœtal and maternal vessels is of course negatived by the anatomical structure of the organ.

The actual facts available regarding the nature of the placental interchanges are exceedingly scanty. From a comparative analysis of the blood delivered to the placenta by the umbilical arteries, and conveyed from it by the vein, we learn that the fœtal blood acquires O, and loses CO_2 in its passage through the placenta. About the nutritional interchanges nothing is known. It is easy to understand that diffusible substances such as water, salt, sugar, peptones, etc., may pass through the dialyser, but whence the fœtus obtains its supplies of indiffusible substances such as fat we do not know. And yet an extensive deposition of fat in the fœtal organism does take place, for Fehling has shown that the proportion of fat to the total weight increases from ·45 per cent at the fourth month of gestation to 9·1 per cent at term. It is extremely unlikely that all this fat comes through the placenta from the maternal blood. The theory has been suggested that it may be carried through by leucocytosis, but leucocytes have never been observed to pass through the villi, and any extensive diapedesis could hardly have escaped observation. The conclusion seems inevitable that the fœtus produces its own fat. The same must be said of the important pigments, such as hæmoglobin, bilirubin, the iris pigment, etc. The attempt has frequently been made to prove by experiment that various gaseous and solid substances, both soluble and insoluble, may pass through the placenta. While the attempt has succeeded with gases and soluble solids, all attempts to pass insoluble solids through the placenta have broken down. It has been held by many that certain micro-organisms, such as streptococci, may pass through, and directly transfer disease from the mother to the fœtus, but even on this point the results obtained are not free from objection. The transference of disease is not open to doubt, but it is questionable whether the organisms themselves pass over, and even if they do so, whether they pass through the placenta.

Fœtal Excretory Functions.—We know very little of the means by which the predigested nutriment conveyed to the fœtus through the umbilical vein is worked up into the various tissues of the body, nor how the waste inseparable from such a process is disposed of. Carbonic acid, the direct product of combustion, is eliminated through the umbilical vein, the fœtal lungs being functionless, and the placenta, therefore, the true respiratory organ of the fœtus. Very early provision is, however, made for excretion by other channels, viz. through the liver, the kidneys, and the skin.

The fœtal liver is a bilobed organ of predominant size as early as the fourth week of gestation in the human embryo. During the second month it increases out of all proportion to the other viscera, causing the abdomen to appear markedly protuberant. In the later months it becomes proportionately smaller than at this period, but remains up to term relatively larger than in the adult. In the mature fœtus it weighs $\frac{1}{18}$th part of the total body weight, while in the adult the proportion is about $\frac{1}{36}$th. The lower border reaches the level of the iliac crest, and the organ occupies one-half of the entire pelvi-abdominal cavity, and contains one-half of the total bulk of blood in the body. From the time of establishment of the allantoic circulation onwards it receives a free supply of pure blood directly from the chorion. The gall-bladder appears during the second month, and in the third month contains a yellow fluid which must be considered to be a true biliary secretion, since it contains both bile salts and acids (Preyer). Bile pigment has been demonstrated at the fifth month. The biliary secretion is the principal source of the meconium, which is present in the upper gut from the time of appearance of bile in the gall-bladder onwards. Another proof of the activity of the liver is to be found in the fact that a large deposit of glycogen is present in the fœtal muscles, and also, though to a less extent, in the amnion and placenta. The presence of this substance in the placenta was first noted by Claude Bernard. It is well recognised that the liver is the source of glycogen formation in the adult, and we are at liberty to assume with safety that the same holds good of the fœtus. Further, the liver is the great heat-producing organ, and we therefore find that the temperature of the fœtus usually exceeds the internal temperature of the maternal body by ·5° to 1° F. Lastly, urea, also a product of the metabolism of the liver, is present in fœtal urine and in the liquor amnii, and has been detected in the latter fluid as early as the sixth week of gestation by Prochownik. It becomes clear

from these facts that from a very early period of gestation onwards the liver is an active organ, with functions similar in all respects to those of the adult organ, and its work as a food transformer, heat producer, and waste eliminator must be of commanding importance in the general nutrition of the fœtus.

The Fœtal Kidneys.—Observations are greatly needed as to the time at which a renal secretion first appears in the fœtus. It is well established that during the last two months of gestation a fluid is commonly present in greater or less amount in the bladder, which since it contains urea, water, albumin, and chlorides, is obviously a true renal secretion. In cases of congenital occlusion of the urethra the bladder is usually found enormously distended; this leads to the conclusion that not only is urine secreted continuously during this period, but, under normal conditions, it is from time to time voided from the bladder into the amniotic sac. Albumin is commonly found in the urine of new-born children whether premature or mature; Preyer has shown that it transudes from the fœtal blood through the glomeruli, and he attributes its presence to the ill-developed state of the glomerular epithelium in the fœtus. It is most abundant about three days after birth, and disappears about the eighth day. It has been shown also that substances administered to the mother will find their way into the fœtal urine and liquor amnii. Thus if benzoate of soda be given to a woman in labour, hippuric acid may be detected in the urine of the new-born child, and also in the liquor amnii. The substance has thus probably passed first through the placenta from the maternal to the fœtal blood, and thence through the kidneys into the urine, and through the skin. or urine into the liquor amnii.

The Skin.—The adult skin contains two sets of glandular structures, the sebaceous glands and the sudoriparous glands. In the fœtus the sebaceous glands appear first, being active as early as the 5th month, when vernix begins to accumulate upon its surface. The sweat glands, although they appear about the same period (5th month) are unprovided with ducts until the 7th month, so that they cannot be functionally active before the latter date. The vernix consists of almost pure fat, with which are mixed debris of epithelial cells and lanugo hairs. The structure of the skin is very simple, the horny layer of the epidermis being practically absent, while the rete is very vascular; it is probable, therefore, that transudation can readily occur through the skin from the fœtal blood to the liquor amnii. In this way urea, *e.g.*, may enter the liquor amnii.

Beyond the abundant production of fat by the sebaceous glands, nothing is, however, definitely known of the functions of the fœtal skin.

Other Fœtal Glands.—The activity of the mammæ at birth has often been noted, but is of no physiological importance. In some mammals a proteolytic ferment has been detected in the fœtal stomach, and an amylolytic ferment in the pancreas, but these observations have not been confirmed in the human fœtus. Regarding the functions of the thymus and thyroid glands in intra-uterine life nothing is known.

Meconium.—This substance is the most important of the fœtal excretions, the others have already been referred to, viz. the urine and the vernix caseosa. Its principal constituent is bile, with which are mixed the secretions, if any, of the gastric and intestinal glands, and extraneous materials, such as lanugo hairs and epidermis scales, which have been carried into the intestinal canal by swallowed liquor amnii.

The meconium is commonly found equally distributed in the mature fœtus, from the duodenum to the rectum. This appears to indicate that peristaltic movements occur in the fœtal gut which pass the meconium mass downwards as it accumulates in the duodenum; it is possible that these peristaltic movements are excited reflexly by the contact of the acid meconium with the mucous membrane. Gas is never found in the fœtal stomach or gut, and no putrefactive processes occur in the meconium. This is a point of forensic importance in relation to the question of infanticide *versus* still-birth. It is curious to note that in the embryo chick fæcal masses are also formed in the intestine before it leaves the shell, which consist largely of products of the hepatic secretion (Preyer).

The Amnion.—The structure of the amnion does not undergo much alteration after its first formation. It consists of a single layer of cuboidal or low columnar epithelium resting upon a stratum of loose connective tissue with wide meshes. It can be easily stripped off the chorion læve and the fœtal surface of the placenta up to the insertion of the umbilical cord, but not from the cord itself. The fluid it contains is probably fœtal in origin; it is formed *ab initio* in the blastodermic vesicle by segregation from the fœtal tissues; after the formation of the placenta it probably comes, in great part, by transudation, from the vessels exposed upon the fœtal surface; during the later months transudation may also occur through the fœtal skin, and small quantities of urine may from time to time be voided from the fœtal bladder. It must, however, be admitted that this view is open to objection, and many observers maintain that it arises from the maternal blood-vessels. In favour of this view are the following facts:—(1) That the fœtus is known to swallow considerable quantities during intra-uterine life, and it seems unlikely that it should excrete fluid merely to be swallowed again; (2) There is experimental evidence that

salts administered to the mother may be detected in the liquor amnii. The value of this observation is, however, somewhat invalidated by the fact that in a proportion of the experiments the salts were also discovered in the fœtal urine, so that they may have reached the liquor amnii, not directly from the maternal vessels, but through the medium of the fœtal circulation. The question must be regarded as unsettled at the present time.

Liquor amnii is a clear, pale fluid resembling a serous fluid in its general characters and composition. Its specific gravity is low (1006 to 1008), its reaction alkaline, and its composition, according to Hoppe-Seyler, is as follows:—

	Per cent.
Water	98·41
Albumin	0·19
Inorganic salts	0·59
Extractives	0·81
	100·00

The amount of albumin varies at different periods of gestation. At the fourth month there is as much as 10·77 per cent (Vogt and Scherer). Gradually the proportion diminishes, until at term it is considerably under 1 per cent, although Hoppe-Seyler's figures are lower than those of most other observers. The most important extractive is *urea*, but of this there is no more than is usually found in serous fluids. Prochownik found it as early as the sixth week of gestation. It probably comes from the fœtal skin and kidneys, at any rate in the later months of gestation. Various matters in suspension, chiefly skin products, such as lanugo hairs, epidermal scales, and masses of vernix caseosa, are also found in it. Its amount at the end of gestation varies greatly, but is seldom more than fifty or less than ten ounces.

The function of the amniotic fluid is mainly protective. It assists in the maintenance of a constant temperature, diminishes the risks of injury from without, equalises pressure, allows of the free movements of the fœtus *in utero*, and during labour cleanses the passages by flushing them from within. When deficient in amount, pressure of the uterus upon the child may lead to the formation of adhesions and resulting deformities. It is probable, also, that it may be swallowed by the fœtus, and thus become a source of water-supply. It is interesting to recall the fact that Harvey observed that the embryo chick swallowed its liquor amnii, and similar observations were made by Haller, but no recent confirmation of this statement is to hand. The presence of lanugo hairs, epidermal scales, and particles of fat in the meconium of the human fœtus is certainly not uncommon, and occasionally hair balls composed of lanugo have been found in the fœtal stomach on post-mortem examination. The presence of these substances in the intestinal tract can only be explained by the assumption that they have been carried thither by the liquor amnii. In any case it can hardly be important as a source of nutriment, but it may well be useful in supplying part of the water required by the fœtal tissues.

The Umbilical Cord.—The development of this structure has been already described. In the earlier months of gestation it is composed of two arteries, two veins, the allantois, the umbilical vesicle with its duct, and the mesoblastic tissues of the ventral stalk. The cœlom or body-cavity is also prolonged into it, and a knuckle of gut may be contained within the cord at its fœtal extremity. The umbilical vesicle soon disappears with its omphalo-mesenteric duct. It is stated that a trace of the umbilical vesicle may be found in the cord at term, in the form of a minute yellowish body, placed at its placental insertion. Then the cœlom closes and the allantois disappears, but traces of the latter in the form of an epithelial tubule may be found in the cord up to the third or fourth month. Then the two veins fuse to form a single channel, the arteries remaining distinct. Sometimes permanent non-closure of the cœlom occurs, giving rise to the condition known as *exomphalos*. The coats of the arteries and veins are of much the same thickness, and both arteries and veins are provided with valves (Kollmann). The substance of the cord consists of a loose connective tissue of gelatinous consistence named Wharton's jelly. This tissue is irregularly disposed round the vessels, making the cord of unequal thickness, and giving rise to protuberances or "false knots" at different parts, which are generally found to contain in addition a loop of the vessels. The epithelial covering of the cord consists of stratified cubical cells in the earlier months, but at term there is usually only a single layer. Torsion of the vessels is evident as early as the third month; the arteries become twisted round the vein, usually from left to right. There is no satisfactory explanation of this occurrence. From exaggerated torsion the cord may be nipped nearly in two; but this is usually now regarded as a post-mortem phenomenon, and not as the cause of death of the fœtus. Its occurrence is confined to aborted ova. The cord varies in length from 5 to 60 inches, the average being from 18 to 24 inches. When very long the fœtus may slip through a loop, causing a *true knot*; the effect of this accident upon the circulation is not serious. In the case of twins with long cords very complicated knotting and interlacement may occur. Loops of the cord are also frequently wound around some part of the body of the fœtus, such as the neck or limbs. According to Braun this may cause strangula-

tion of a limb, and may even cut through the neck as far as the vertebral column, but never causes amputation. The cord is usually inserted into the placenta at or about its centre. It may, however, be placed near the margin (*excentric insertion*), or upon the edge (*battledore insertion*), or into the membranes, where its vessels divide into branches running to the placenta (*velamentous insertion*). These irregularities occur when the placenta is not equally developed around the insertion of the primary ventral stalk, *i.e.* the spot at which the allantoic vessels just reach the wall of the ovum.

Determination of the Period of Development.—During the first six or seven weeks of its development the human embryo is indistinguishable, except by an expert embryologist, from that of birds and other mammals. An embryo, measuring about 7 mm. in length, was actually described in great detail in the *Archiv für Anatomie und Gynecologie*, 1876, by Krause as a human embryo of four weeks' development, the error being exposed several years later by the anatomist His. About the end of the second month definite characteristics appear which serve to distinguish it from the embryos of other creatures. It is usual to speak of the *embryo* during the first two months, and of the *fœtus* subsequent to that period.

The Human Embryo.—Aborted ova of the first two months of gestation frequently show no trace of the embryo. If the ovum sac has ruptured, the embryo escapes with the liquor amnii, and so is lost; but in unruptured ova it is often not found, its disappearance being then due to the disintegration and absorption which rapidly follow upon the death of the embryo at this early period. Under such circumstances the age of the ovum must be estimated from the size and characters of the membranes.

The following table gives the diameters of the sac of the ovum at different periods of the first three months of gestation:—

1 week	. .	3·7 × 4 mm.	Leopold.
2 weeks	. .	12 × 14 mm.	do.
3 ,,	. .	25 mm.	His.
8 ,,	. .	4·5 cm.	Galabin.
12 ,,	. .	10 cm.	do.

From the second week to the end of the second month the chorion is found covered in all parts with chorionic villi. During the third month the placenta is more or less clearly delineated by the disappearance of the villi over the general chorionic surface. The umbilical cord is usually found in whole or part after the end of the first month. During the first three months aborted ova are usually discharged from the uterus without any decidual investiture. The attachments of the fœtal to the maternal structures at this period are so delicate that they are easily ruptured by the uterine contractions, and the chorionic sac is thus discharged without its decidual coverings, which usually follow later. When the placenta is formed the fœtal attachments are much firmer, and shreds or strips of decidual are usually found adhering to the chorion.

The earliest human embryo which has been accurately described belonged to the end of the second week, and measured 2·2 mm. in length (His). The same observer estimates that by the end of the third week it measures 4 mm., and by the end of the fourth week 7 to 7·5 mm. At this period the cephalic and caudal extremities are bent towards the ventral aspect, and the above measurements represent only the long diameter of the flexed embryo, *i.e.* the cephalo-pelvic diameter; straightened out it is, of course, much longer. Other points deserving of notice in embryos of the first month are:—(1) The amnion is closely apposed to the dorsal surface, *i.e.* there is very little liquor amnii. (2) Attached to the ventral aspect are the umbilical vesicle, a pediculated sac measuring 2 to 3 mm. in diameter, and a very short umbilical cord. (3) The ocular and auditory vesicles are apparent at *the fourth week*, and the limbs are represented by small unsegmented lateral buds. During the second month the embryo grows from 8 mm. in length at its beginning to about 20 mm. at its close. During this period marked changes have occurred, which render it easily recognisable as a human embryo. The ventral aspect becomes closed, leading to the disappearance of the umbilical vesicle, and the umbilical cord becomes a prominent structure, lying just above the curved tail-end of the embryo. The abdomen is protuberant owing to the rapid growth of the liver, which even at this early period predominates greatly in size over the other viscera, and the trunk in consequence exceeds the head in girth; after the eighth week the proportion becomes reversed owing to the rapid increase of the size of the head. At this period eyes, nose, mouth, and ears are formed, and the shape and characters of the pinna are in themselves distinctive of the human species. The limbs are segmented, and the fingers and toes indicated by rows of buds of fairly equal size. The formation of a distinct neck by closure of the lower branchial arches also occurs at the end of the second month. The genital cleft appears, but the external sexual characters are undifferentiated.

During the third month the fœtus increases to the length of 7 to 9 cm. The marked increase in length during this period is more apparent than real, for it can now be measured from vertex to heel, whereas previously it was measured only from vertex to pelvis. The umbilical cord is now as long as the fœtus, and becomes twisted. The external sexual organs are still undifferentiated, but the presence or

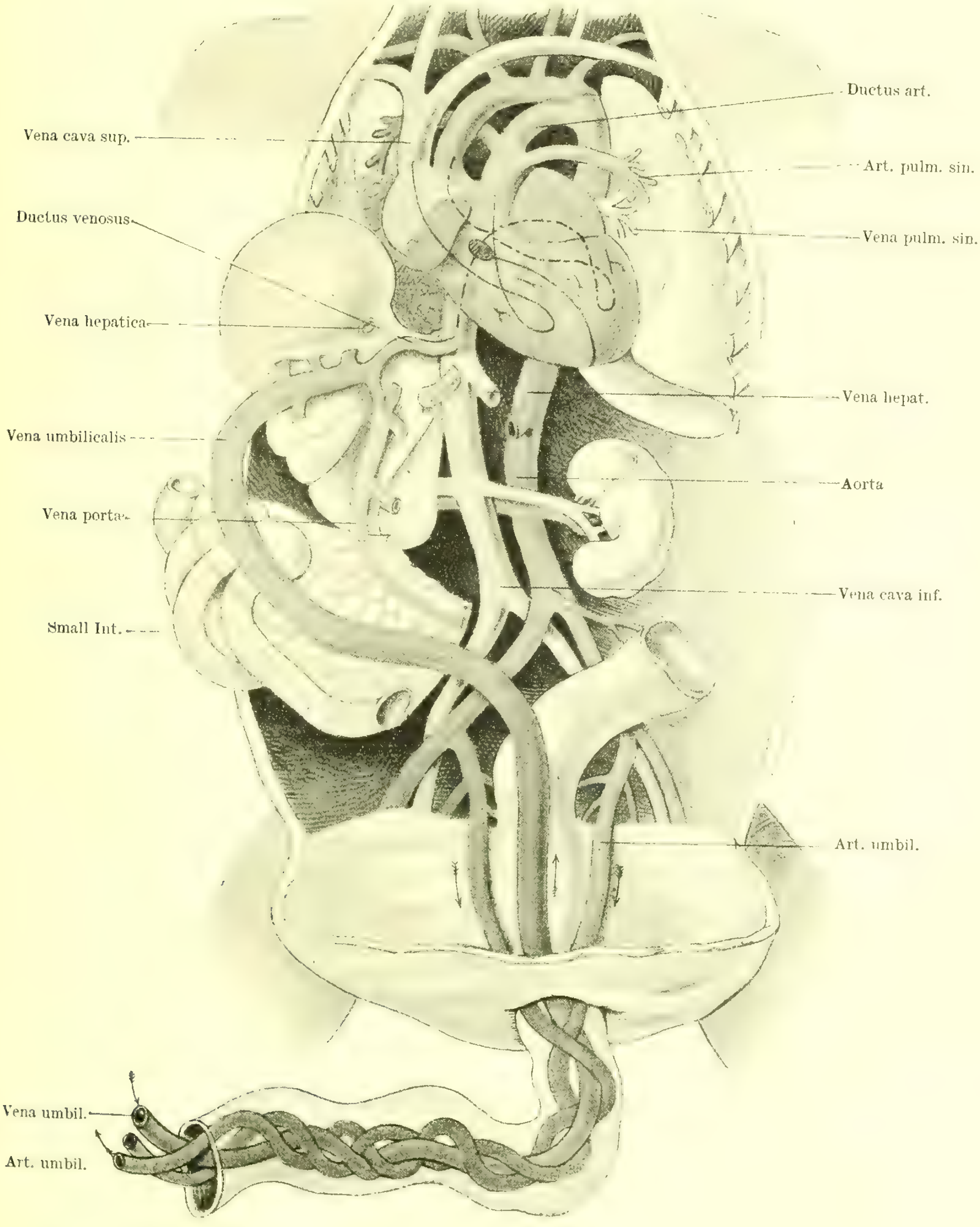
absence of the uterus may be made out by dissection. The rate of growth of the fœtus, both in length and weight, is subject to wide variations during the remaining months of gestation. Thus the length, as stated by Haecker, at the end of the fourth month may be as small as 10 cm. or as great as 17 cm. The rate of growth of the placenta is almost equally variable; its thickness at the end of the fourth month is, however, said to be fairly definite, viz. 10 mm. The following table gives the average length of the fœtus at different periods, and its average weight during the later months:—

Period.	Length.	Weight.
2 weeks	2·2 mm.	
3 ,,	4 mm.	
4 ,,	7·5 mm.	
2 months (lunar)	20 mm.	
3 ,,	7 to 9 cm.	
4 ,,	10 to 17 cm.	
5 ,,	18 to 27 cm.	$\frac{1}{2}$ lb.
6 ,,	28 to 34 cm.	$1\frac{1}{2}$ lbs.
7 ,,	35 to 38 cm.	$2\frac{1}{2}$,,
8 ,,	39 to 41 cm.	$3\frac{1}{2}$,,
9 ,,	42 to 44 cm.	$4\frac{1}{4}$,,
10 ,,	50 to 53 cm.	6 to 9 ,, (Average, 7-$7\frac{1}{2}$ lbs.)

The Mature Fœtus.—The points by which a mature may be distinguished from a premature fœtus are of some practical interest, and may be of importance from a forensic standpoint.

Length and weight are subject to wide variations. The average length varies from 19 to 23 inches; a fœtus measuring 20 inches may be safely assumed to be mature, but the converse is untenable, since the mature fœtus may be much shorter than this. A premature fœtus, however, probably never attains a length of 20 inches. The average weight is subject to still wider variations, and may be stated at from 6 to 9 lbs. A healthy fœtus weighing 6 lbs. is probably mature, but here again the converse is untenable; it may be mature, and yet weigh less than this. Premature children may exceed this weight from the presence of disease, such as ascites or abdominal tumours, syphilitic disease of the liver, etc. The skin of the mature fœtus is plump and smooth, presenting a marked contrast to its lax and wrinkled condition in a fœtus from about the seventh month of gestation, the difference being due to the formation in the last two months of an abundant deposit of subcutaneous fat. The lanugo has disappeared from all parts, except perhaps a trace upon the cheeks; the hair on the scalp is about an inch long, and the eyebrows and eyelashes are well developed. The nails project slightly beyond the tips of the fingers and toes. The skull bones are approximated except at the fontanelles, and if a dissection can be made centres of ossification will be found in the lower femoral epiphysis, the astragalus, and the cuboid bones. The breasts are plump in both sexes, and a little secretion can often be expressed, which on microscopic examination is found to contain colostrum corpuscles and fat globules. If not asphyxiated, the fœtus moves and cries vigorously when born, thus presenting a marked contrast to the torpor of a prematurely born child; it also passes meconium and urine within a few hours of birth.

The Fœtal Circulation.—The umbilical vein which brings arterial blood from the placenta enters the trunk at the umbilicus and runs along the anterior abdominal wall to reach the lower surface of the liver. Here it gives off twigs to the left lobe, the lobus quadratus, and lobus Spigelii, which thus receive a direct supply of pure arterial blood. It then divides into two main branches, one of which enters the portal vein, and the other, called the ductus venosus, joins the inferior vena cava. It will thus be seen that with the exception of the venous blood brought from the alimentary canal by the portal vein, none but pure arterial blood enters the liver. No other fœtal organ receives anything like so large a supply of aerated blood. From the liver the blood is carried through the hepatic veins to the inferior vena cava. The blood in this large trunk is mixed; venous blood coming from the pelvis and lower extremities by the iliac veins, and from the liver by the hepatic veins, while arterial blood comes from the ductus venosus. Entering the floor of the right auricle the blood-stream is directed by the Eustachian valve across the chamber to the foramen ovale, through which it passes *in toto* into the left auricle. Thence it flows through the mitral valve into the left ventricle, and thence into the aorta, to be distributed in greater part through the innominate, left carotid, and left subclavian trunks to the head, neck, and upper extremities. These parts, therefore, receive the purest arterial blood next to the liver, and further, they receive practically all the blood which has already passed through the liver. From the head, neck, and upper extremities the venous blood is conveyed through the superior vena cava to the right auricle, and thence through the tricuspid valve into the right ventricle; it is believed that there is little or no admixture of the two blood-streams which cross one another in the right auricle. From the right ventricle the blood passes into the pulmonary artery, which, after giving off small branches to the lungs, passes, as the ductus arteriosus, into the descending portion of the thoracic aorta. It will be observed that the blood entering the aorta at this point is venous blood from the head and neck, etc. Probably a little mixed blood comes down from the left ventricle as well, but there can be only a small proportion of arterial blood contained in the



Fœtal Circulation.

abdominal aorta. Part of this blood passes to the abdominal viscera, the pelvis and lower extremities, the greater part is carried by the internal iliac arteries into the two hypogastric arteries, which pass up the anterior abdominal wall to the umbilicus, where they leave the body and enter the umbilical cord. They are now known as the umbilical arteries, and pass, coiling round the umbilical vein, to the fœtal surface of the placenta.

The Changes in the Fœtal Circulation at Birth.—These are due to two causes: first, the expansion of the lungs by respiration; and second, the separation of the fœtus from its placenta by division of the cord. The immediate effect of expansion of the lungs is to divert a great part of the blood in the pulmonary artery to the lungs; as a result the ductus arteriosus becomes greatly contracted. The immediate effect of division of the cord is to reduce the pressure in the right auricle by diminishing greatly the amount of blood brought to it by the inferior vena cava. At the same time the pressure in the left auricle is raised, because the inflow through the pulmonary veins is augmented by the increased amount of blood now flowing through the lungs. As a result the pressure in the two auricles is equalised, and when they contract simultaneously in systole the flap valve of the foramen ovale becomes closed, and the passage of blood from right to left auricle is arrested. Practically all the blood entering the right auricle is now passed through the tricuspid valve into the right ventricle and thence to the lungs. The sudden reduction of the blood-pressure in the right heart probably also assists the closure of the ductus arteriosus. The umbilical vein contracts as there is now no blood entering it from the placenta, and along with the ductus venosus it becomes obliterated without thrombosis. In the adult the former persists as a fibrous cord enclosed in a fold of peritoneum, being known as the falciform ligament of the liver. The umbilical arteries and ductus arteriosus are also said to close without thrombosis, although the presumption would be that as some blood is able to enter these vessels, clotting would occur. The foramen ovale is usually completely closed by the eighth to tenth day after birth; sometimes it remains permanently patent, giving rise to the condition known as *morbus ceruleus.* The ductus arteriosus usually closes rather earlier than the foramen ovale, being converted into the ligamentum arteriosum.

Disposition of the Fœtus in Utero.—Sectional anatomy has added some valuable facts to our knowledge of this subject within recent years. The general arrangement of the fœtal parts is one of moderate flexion; as a result the body of the fœtus forms a compact ovoid, the long diameter of which measures about 10 inches, *i.e.* rather less than half the total length of the body. The advantage of this arrangement is sufficiently obvious. The head is usually found to be flexed, the chin touching the sternum, before the onset of labour, a point which will be again considered in its bearing upon the mechanism of labour. Frequently, also, the head is rotated a little to one or other side, and bent towards the shoulder. Minor deviations from this arrangement may be met with, such as extension of the legs, but they are comparatively unimportant. Frequent changes in the relation of the long axis of the fœtal ovoid to that of the uterus occur during the last weeks of pregnancy.

Fog. *See* Meteorology (*Effect of Fog on Health*).

Foie Silex.—The "flint liver" which is found in cases of congenital syphilis. *See* Liver, Diseases of (*Hereditary Syphilis*).

Fold.—A term commonly used in Anatomy and Embryology for the doubling or bending of a structure on itself.

Folie Circulaire. *See* Insanity, Nature and Symptoms (*Alternating Insanity*).

Folie du Doute. *See* Alcoholic Insanity (*Folie du Doute*); Insanity, Nature and Symptoms (*Defects of Inhibition, Swithering Insanity*).

Follicle.—A glandular sac or crypt, *e.g.* a Graafian follicle in the ovary, a Lieberkühnian follicle of the intestine, and an agminated follicle of the ileum; *folliculitis* is inflammation of such follicles, but more particularly of the hair follicles of the skin. *See* Alopecia; Acne; Skin, Tuberculosis (*Lichen Scrofulosorum*).

Fomentation.—*Fomentations* or *fomenta* are cloths or sponges wrung out in hot water (often containing a sedative or stimulating medicine) and applied to the skin over the seat of an injury or a disease; they have been recommended also in labour, in inertia uteri; when turpentine is sprinkled on the cloth or flannel a turpentine *stupe* is prepared, and when a bag of hot salt or bran is used it is called a "dry" fomentation.

Fomites.—The plural of *fomes,* which literally means fuel, and is employed, in medicine, for the various substances or materials (*e.g.* clothes, curtains, letters, etc.) which may serve to carry the germs of disease from one person to another.

Fontanelles.—The membranous interspaces between the bones of the head of the fœtus and new-born infant which allow changes in the outline of the cranium to occur (moulding) without prejudicial effects; their recognition is important in the diagnosis of the presentation and position of the child in labour

See Bregma; Children, Clinical Examination of (*The Head*); Hydrocephalus; Labour, Physiology (*Passenger*, *Fœtus*); Meningitis, Tuberculous and Posterior Basic (*Symptoms*, *Fulness of Anterior Fontanelle*); Rickets (*Individual Symptoms*, *Head*, *Craniotabes*); Spina Bifida (*Complications*, *Bulging of Fontanelle*).

Food.

See also Allantiasis; Appetite; Asthma (*Treatment*, *Diet*); Atrophy, Infantile (*Improper Feeding*); Diet; Digestion and Metabolism; Feeding, Forcible; Gastro-Intestinal Disorders of Infancy; Infant Feeding; Intestines, Diseases of (*Enteritis*); Invalid Feeding; Milk; Physiology, Food and Digestion; Stomach and Duodenum, Diseases of (*General Etiology*, *Food*); Scurvy, Infantile (*Causes*); Scurvy in Adults (*Causes*); Toxicology (*Poisoning by Copper*, *Lead*, *and Food Stuffs*); Trades, Dangerous (*Lead Poisoning*).

I. Characteristics of Good Meat

In all cases the carcase should be that of a well-nourished animal, without signs of wasting or attenuation; the pleura and peritoneum should be free from adhesions or staining. Good meat is firm and elastic to the touch, without œdema or emphysema, not pitting nor crackling on pressure; it should be juicy, but not wet nor flabby; the colour should be uniform, without brown or discoloured patches. Good beef is of a bright red colour, marbled with fat; veal is always paler and less firm to the touch; mutton is dullish red, firm, the fat hard and usually white; in both beef and mutton a uniform yellowness of the carcase, almost a saffron tint, may be associated with perfectly healthy conditions. The carcase of the pig should be plump; the flesh is naturally pale and the fat somewhat soft; the skin should not set in folds or wrinkles, and should be without stains or blotches; bruises and scratches are not uncommon. In all cases when sufficient time has elapsed for the carcase to cool and set, the fat should be firm and the suet hard, containing no watery jelly or juice, free from blood-stains, and creamy white to yellowish in colour. Particular attention should always be paid to the connective tissue about the flanks, shoulders, and diaphragm and region of the kidneys; signs of wetness, œdema, imperfect setting, or diseased glands should be absent; the thoracic and abdominal parietes should be without signs of old adhesions, staining, or evidence that anything has been stripped away, which is sometimes done to remove signs of inflammatory disease. The odour should be sweet and agreeable; a skewer thrust deeply into the flesh should have no unpleasant odour when withdrawn. Bull beef, it must be remembered, is usually dark, but in other cases abnormal darkness of the flesh is to be regarded as suspicious of imperfect bleeding.

The dressing should always be completed with as little delay as possible immediately after the animal has been killed. In the case of cattle, the animal, immediately it is knocked down, should be bled. Every few seconds which the blood remains in it is a matter of consequence, and tends to cause deterioration of the flesh. The advantage of the Jewish method of slaughtering cattle, namely, by cutting the throat, is that the blood does not remain in the carcase at all after death. The viscera, especially the abdominal viscera, should be removed as soon as possible, or staining will result. The appearance of the carcase is dependent to a considerable extent upon the manner in which it is dressed. A good butcher, clean, careful, and prompt in the dressing, will secure a better wholesale price than an indifferent one. Care should be taken to keep the carcase clean during the process, so as to lessen or prevent the necessity for washing. All badly dressed carcases should be regarded with suspicion, since diseased animals are frequently dressed hurriedly, and by unskilled hands, and present a slovenly appearance. But it must be remembered that some parts of the kingdom are noted for the bad dressing of the carcases, which are often packed and despatched to market before they have had time to cool and set, and carcases of calves are imported from Holland with their hides on and the dressing roughly and imperfectly done.

The age of the animal will be recognised by the degree of ossification and the diminished amount of cartilage; older animals have less fat, and the flesh becomes firmer and harder. Still-born or newly-born calves have a watery appearance of flesh, the fat is tallow-like, and the hoofs yellow and soft, readily indented with the nail.

Wherever possible, the viscera should be examined; in routine examination of meat it is essential that this should be done.

Refrigerated meat is imported into this country in immense quantities, and usually in prime condition, and it keeps well on exposure. Beef is imported in quarters wrapped in muslin cloths; the fat is sometimes stained with the meat juice, which gives it a dullish red appearance. Carcases of sheep are imported entire, usually wrapped in muslin cloths. When the thawing process sets in, it commences on the outside; consequently the appearance of such meat may be uninviting on account of the wetness on the surface, but this wetness must not be confounded with the œdema of diseased meat, which is a very different condition.

Frozen rabbits and frozen poultry have recently been imported in very large quantities, cargoes of many tons arriving in perfectly sound condition, in the case of rabbits from Australia and New Zealand, and in the case of poultry, chiefly from Canada.

Characteristics of Unsoundness

In *chronic* disease the carcase is usually more or less emaciated, sometimes to an extreme degree. The flesh of diseased animals is pallid in appearance, and together with the connective tissue may be infiltrated with serum; the fat and visceral connective tissue are also wet and flabby, and the fat will not set; occasionally the pleura is found to have been stripped off from the ribs to remove evidence of pulmonary disease. In *acute* inflammatory diseases the affected organ will present the ordinary signs of inflammation, but if the animal be slaughtered at an early stage of the disease, and is properly bled and dressed, the flesh is usually normal and sound; if, however, the animal has not been killed until nearly moribund, or if the disease has made progress, the carcase will be found to be red and congested from imperfect bleeding; it does not set properly, the flesh is dark, dry, and sticky, and frequently giving off an unwholesome odour of drugs, which can be best detected by plunging a skewer deeply into the flesh and noting the smell upon withdrawal.

Some important conditions affect the wholesomeness of the flesh of animals used for food and have to be considered.

Tuberculosis. — Cattle, pigs, poultry, and rarely sheep suffer from this disease. Cattle, more especially milch cows confined in dairies, are most prone to contract it. In early stages of the disease the flesh of the affected animal is normal and fit for food; as the malady progresses emaciation sets in, the carcase after slaughter is soft, skinny, and dropsical, the fat wet and flabby and presenting all the appearances of unsoundness. The Local Government Board in England have adopted the following recommendations of the Royal Commission on Tuberculosis as a guide to meat inspectors in the inspection of tuberculous carcases of cattle:—

(*a*) When there is miliary tuberculosis of both lungs (*b*) When tuberculous lesions are present on the pleura and peritoneum (*c*) When tuberculous lesions are present in the muscular system or in the lymphatic glands embedded in or between the muscles (*d*) When tuberculous lesions exist in any part of an emaciated carcase	The entire carcase and all the organs may be seized.

(*a*) When the lesions are confined to the lungs and the thoracic lymphatic glands (*b*) When the lesions are confined to the liver (*c*) When the lesions are confined to the pharyngeal lymphatic glands (*d*) When the lesions are confined to any combination of the foregoing, but are collectively small in extent	The carcase, if otherwise healthy, shall not be condemned, but every part of it containing tuberculous lesions shall be seized.

In view of the greater tendency to generalisation of tuberculosis in the pig, we consider that the presence of tubercular deposit in any degree should involve seizure of the whole carcase and of the organs.

In respect of foreign dead meat, seizure shall ensue in every case where the pleura have been "stripped."

Amongst acute diseases, *pleuro-pneumonia* of the acute character is one of the most important. The usual pathological signs are met with in the lungs, and the extent to which the carcase is prejudiced in respect to its fitness for human food will depend upon the degree to which the disease has advanced before the animal is slaughtered.

Anthrax and *anthracic diseases* affect cattle, sheep, and sometimes pigs. The disease is rapidly fatal, and the local lesions in the glands, liver, and spleen develop rapidly, the flesh assuming a peculiar odour, and decomposing much sooner than usual. The carcase in every instance is unfit for human food and should be destroyed.

"*Braxy*" is a term applied to a variety of conditions widely divergent. Wet braxy appears to include dropsical conditions in the sheep, red braxy is a term sometimes given in inflammatory and abnormal parturient conditions. The term also appears sometimes to be used as a synonym for anthrax. Under no circumstances should the flesh of an animal dying from anthrax or any allied disease be allowed to pass into the market.

Foot and mouth disease is common amongst cattle, sheep, and pigs, and is associated with ulceration of the tongue and mucous membrane of the mouth, and with the formation of blisters around the hoofs. The disease is very infectious, but unless aggravated to an extent which has prevented the animal feeding, the flesh is not affected, and presents the appearance of normal flesh, and may be safely used for food.

Swine fever, known also as "purples," "swine typhoid, "soldier," etc., is a very acute and fatal contagious disease, which renders the flesh of the affected animal unfit for food. Red patches or blotches appear on the skin, the redness extending in a marked degree through the subcutaneous fat down to the flesh; emaciation

is rapid, and the flesh becomes pale, flaccid, and dropsical, and of a peculiar and unwholesome odour. The intestines, especially the large intestine, exhibit lesions which bear a resemblance to those of typhoid fever in man.

Flesh from Animals which have died, or which have been damaged or killed by Accident.—The carcases of animals drowned or smothered or naturally dead are unbled and dark, abdominal parietes are stained, and signs of decomposition usually evident; such carcases should be condemned. In cases of fractures, wounds, and bruises, if the animal be promptly slaughtered and bled, the damaged portions only should be condemned; much depends on the extent of the injuries.

Parasitic diseases by which the human being may be attacked are of great importance. Two at least may be received through the medium of pork, viz. the *Tæniadæ* and the *Trichina spiralis.*

The encysted state of the Tænia solium of man constitutes the *Cysticercus cellulosæ* which commonly affects the pig, in which it gives rise to the disease known as "measles." The "measly" pork contains the scolex in its cyst; the cysts average the size of a small pea, and are found chiefly in the muscular tissues embedded between the fibres, voluntary or involuntary, as well as in various organs; they are not met with in the fat. The parasite is difficult or impossible to detect during life, but is readily visible when the animal is killed and opened. The carcase should be condemned and destroyed. Hams and bacon containing the remains of the parasite should be similarly dealt with.

Trichina spiralis is a small thread-like worm coiled in minute ovoid cysts *within* the muscular fibres, and is found usually in the pig. Each cyst contains one immature trichina which is liberated when the capsule is dissolved by the processes of digestion. The liberated trichinæ develop rapidly, and give rise to the disease known as trichinosis. Careful examination is necessary for the detection of the trichinæ; the pork appears speckled, and if a thin section be immersed in liquor potassæ for a few minutes, and the translucent section examined with a lens, the coiled-up worm will be seen. All the flesh of an infected animal should be destroyed.

Distoma hepaticum or fluke is commonly found in the liver of the sheep; a few of these parasites, which are sole-shaped, and from half an inch to one inch in length, are usually found in the liver of otherwise perfectly sound sheep, and exercise no prejudicial effect whatever upon the flesh, but as their numbers increase the results of pressure and obstruction to the flow of bile ensue; jaundice, dropsy, diarrhœa, loss of hair, and emaciation set in rapidly, and "rot" results. The disease is met with in all parts of the country, but more especially in damp and wet localities, and during the long-continued prevalence of wet weather; the eggs and the embryo are developed in water, and hence wet seasons encourage the spread of the disease.

The carcase should be destroyed if it is deteriorated, and the liver should also be destroyed.

Blown Veal and Lamb.—In many towns the practice still exists among the lower class of butchers, of blowing up, with the breath, the connective tissue of veal and lamb, and thereby giving an appearance of plumpness to poor meat; this disgusting fraud is completed by taking melted fat into the mouth and blowing it over the freshly-dressed carcase. The practice is an offence against ordinary by-laws, and may be recognised by the emphysematous condition of meat which has been subjected to it.

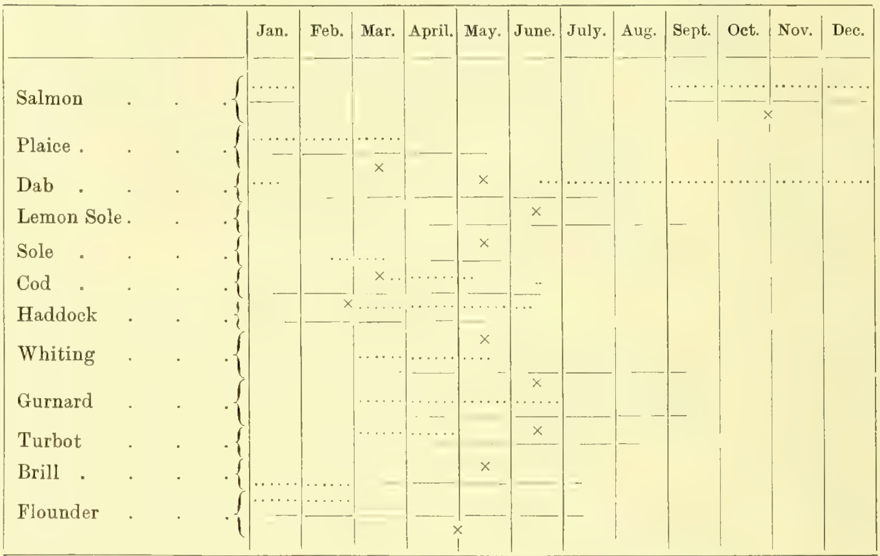
II. Fish

Fish, either fresh or preserved, and shell-fish form an important share of the ordinary food-supply of families, some varieties being as nutritious as flesh foods and more easily digested. Immense quantities of fish are preserved in various ways and imported from distant places.

The chief points to be noted in determining the wholesomeness of edible fresh fish are: (α) the freshness; (β) the season in which they are on the market, as this is associated with their condition as to spawning; (γ) the places from which they are taken, as the nature of their food is involved. With regard to (α) the freshness, fish should be firm to the touch; the duration of the stiffness of recently caught fish depends upon the weather and passes off in from six to twelve hours; the eyes and skin should be bright and glistening, the gills bright red, unless dulled by ice; no trace of offensive smell. Fish keep best if killed and gutted immediately they are caught; if dealt with in this way they remain fresh from two to three days longer; they deteriorate most quickly when caught by the gills in nets and allowed to die slowly in the water. Trawling boats going very long distances to the fisheries are provided with chilling apparatus or ice, by means of which the fish can be brought in good condition from distant fishing grounds. In regard to (β), the influence of season, most varieties of fish are decidedly out of condition during spawning, some also during the time preceding and following it; they become thin and lose their flavour. The time of spawning varies considerably; in the case of salmon, rivers are closed under statutory regulations from five to six months, usually from September to February, with a view as much to "preserve" the fish as to ensure wholesomeness; this close season varies in different districts, and smuggled salmon may be perfectly wholesome.

The following table indicates the spawning periods of the principal edible fishes; the black lines indicate the limits of the spawning period,

the crosses (×) indicate the maximum of spawning; the dotted lines include the months when the fish is "out of season"; this is to some extent a matter of individual opinion, and there are many degrees of being out of *condition*. All of the periods are liable to some slight amount of local or annual variation, but the table may be taken as a fair average :—



Professors Herdman and Boyce have recently published the results of their investigations into oysters and disease, and as an outcome of their experiments in regard to the typhoid bacilli they found by washing infected oysters in a stream of clean sea-water that there was a great diminution or total disappearance of the typhoid bacilli in from one to seven days. Their experiments showed that sea-water was inimical to the growth of the typhoid bacilli, and although their presence was demonstrated in one case on the twenty-first day after addition of the water, still there appears to be no initial or subsequent multiplication of the bacilli.

They also found that the colon group of bacilli is frequently found in shell-fish as sold in towns, and especially in the oyster, but they found no evidence that it occurs in mollusca living in pure sea-water.

They also found the frequent presence in various shell-fish of anaerobic spore-bearing bacilli, showing the characteristics of the bacillus enteritidis sporogenes.

They remark that "it is evident, from these experiments and a consideration of all the facts brought to light in recent years in regard to the bacteriology of shell-fish and its influence on public health, that we must regard oysters, mussels, cockles, etc., as nutritious food matters, which, from their nature and circumstances of their cultivation and sale, are liable to become contaminated with organisms, pathogenic or otherwise, and their deleterious products."

It is unnecessary to insist upon the absolute importance of steps being taken to remove any possible suspicion of sewage contamination from the beds and layings from which oysters are supplied to the market.

With regard to the green oysters which are largely imported from America and laid down

The locality (γ) is chiefly of importance in the case of stationary shell-fish, such as oysters, mussels, etc., taken from sewage-contaminated waters, the noxious matter being sometimes taken in by the fish with its food, or the body of the fish being merely soiled with the filth.

An exceedingly important inquiry was recently made by Dr. Bulstrode into the conditions under which oysters and certain other shell-fish are cultivated and stored along the coast of England and Wales. His investigation is given in detail in the 24th Annual Report to the Local Government Board.

Oysters are perhaps better known than other shell-fish to be possible agents in disseminating typhoid fever and forms of choleraic disease, probably because that class being a more important article of food than other shell-fish, greater attention has been paid to it, but there can be no doubt at all that what applies to oysters also applies to other shell-fish used for food.

Dr. Bulstrode's report makes it abundantly plain that many of the beds in which natives are grown, or foreign oysters are deposited for fattening purposes, are so situated as to be almost necessarily bathed with the effluent of sewers, and the shell-fish are in consequence liable to be fouled with the excreta of persons suffering from diseases of the type of cholera and typhoid fever. The transmission of typhoid by this means is now generally acknowledged.

on various parts of the coast for fattening, the conclusion of Herdman and Boyce is that the green colour is due to copper. In the case of the Marennes oyster the presence of greenness is due to a pigment not associated with copper.

III. Milk and Milk Products

Milk is an exceedingly important article of food. Breast milk is, or at all events ought to be, the sole food in the earlier months of life; cows' milk is used to a greater or less extent throughout the whole of life, and is largely relied upon in many forms of illness. The quantity of cows' milk consumed by a community is evidence of its importance as an article of food. About 24,000 gallons are supplied every day to the city of Liverpool, representing a daily allowance of about one-third of a pint per head of the population. Rather more than half that amount, say approximately 12,500 gallons, is supplied from cows kept in cowsheds within the city boundaries; the remainder, viz. about 11,500 gallons, is imported from various farms in the neighbouring counties. Probably most towns in the kingdom are similarly circumstanced

In order to ensure that the milk shall be wholesome, it is a first essential that the cow from which it is taken shall be healthy, and that its surroundings are healthy, the shippon or byre having adequate space, from at least 600 to 800 cubic feet per cow, being also well lighted, well ventilated, and kept clean in every detail. If these precautions are not rigorously observed, the cow is extremely liable to tuberculosis, commonly of the lungs, as in the case of human beings, but besides the lungs, certain glands, notably the udder, may be affected.

It is gratifying to note that throughout the whole country, but more especially in towns where the supervision which is exercised is more efficient, there is a general improvement in the condition of the places where cows are kept.

When the udder of the cow is affected with tuberculosis, the tubercle bacilli which are breeding there flow away with the milk, and, still retaining all their activity and virulence, may on entering the stomach infect with tuberculosis the person consuming the milk.

The real preventive remedy clearly is to aim at the root of the evil, and (1st) to have the shippons, or cowsheds, or byres so well constructed and kept that the cows shall remain under conditions most favourable to health; and (2nd) to have the cows examined from time to time at appropriate intervals, in order to ascertain that they remain healthy. An additional safeguard, which is in the power of everybody, is to boil the milk for a few seconds, or to sterilise it by raising it to a temperature somewhat below boiling-point, either of which would effectually destroy all tubercle bacilli; the sterilisation can be readily accomplished if the vessel containing the milk is placed in a saucepan or other suitable vessel containing water, which can be heated to boiling.

The question has been mooted as to whether or not, in the event of a cow being so dangerously affected with tuberculosis as to contaminate the milk, compensation should be paid to the owner if he removes it. It must be remembered that no cow-keeper who wilfully keeps a diseased animal upon his premises would have any claim to sympathy or consideration. No man has a right to sell an article which damages the health of the consumer, without duly warning the consumer of the danger that he runs. The consumer is not prepared to meet the risk in the case of milk; moreover, there is no reason why the cows should suffer at all from tuberculosis; in good and sanitary shippons or cowsheds such a condition is uncommon; it is only in those which are badly constructed and badly kept that tuberculosis is frequent.

In contrasting the relative purity of town and country supplies, it is satisfactory to find that adulteration is not frequent in either case. In both town and country milk, however, adulteration with water is found a little more frequently in samples taken on Sundays than on other days. A most important difference is found in the frequency of the presence of the tubercle bacillus in town milk and country milk. It would naturally be thought that, with the very great natural advantages which the country possesses, tubercle would be less frequent than it is in the town. This, however, is very far from being the case, and it is a most serious reflection upon the sanitation of country shippons that tubercle is found more than twice as often in samples from the country as it is in samples from the town: thus, out of 398 town samples (taken from shippons in Liverpool), 20, or 5 per cent, were found to be infected with tubercle, but out of 195 samples taken at the railway stations on arrival from the country, 23, or 11·8 per cent, were found to be infected. This shows the great need for sanitation of country shippons, and that the consumer should take town milk when he can get it, rather than that sent in from the country, where little or no supervision is exercised.

Some valuable information upon the subject of tuberculosis and milk was laid before the two Royal Commissions which were appointed to inquire into the effect on human health of food derived from tuberculous animals: the circumstances determining danger from milk were gone into at considerable length, and the conclusions arrived at are important, and may be held to rest upon the most recent knowledge upon that branch of the subject.

The Report of the Royal Commission, April 1895 (paragraph 60), states:— "According to our experience, then, the condition required for ensuring to the milk of tuberculous cows the

ability to produce tuberculosis in the consumers of their milk, is tuberculous disease of the cow affecting the udder. It should be noted that this affection of the udder is not peculiar to tuberculosis in an advanced stage, but may be found also in mild cases." This paragraph is a somewhat guarded one, and is not at variance with the conclusions from recent investigations, which point to the possibility that a tuberculous animal may give tuberculous milk even if the udder itself is not the seat of disease.

Under the Dairies, Cowsheds, and Milkshops Order of 1899, the Local Government Board prohibit the sale for human food of milk from any cow certified by a veterinary surgeon to be suffering from tuberculous disease of the udder, and provide that such milk shall not be used for the food of swine until it has been boiled.

With regard to ascertaining the presence or absence of the tubercle bacillus in milk, the only dependable means is that of inoculation. The guinea-pig is the animal commonly used, the injection being subcutaneous or peritoneal. The lesions which are the result of such inoculation can be clearly recognised from those which might have been produced accidentally in some other way. When proper precautions are taken, the injection of material free from tubercle bacillus will not produce lesions which can be mistaken for tuberculosis.

The microscopic method alone is unsuitable, because even if tubercle bacilli were equally distributed through tuberculous milk (which they are not), a very large number of them would have to be present before one could expect to find one bacillus now and again in the few drops of milk used for a microscopical examination, *e.g.* to examine one ounce of milk, at least 500 microscopical examinations would be necessary, each one taking twenty minutes to half an hour, and if there were, say, 100 bacilli in that ounce, five such examinations would be necessary before it could be expected that one bacillus could be discovered, yet one bacillus in each drop would represent 10,000 bacilli in a pint of milk.

In inoculating, due care must be taken to ascertain that the animal is perfectly healthy before inoculation, and that it is kept under healthy surroundings in an effectively ventilated animal house; the animal being isolated as soon as any abnormal symptom develops.

Turning to another direction apart altogether from tuberculosis, inoculations were found to be extremely useful in investigating the relationship between milk and infantile diarrhœa; it has been found—in the summer and autumn at least—that in some cases when the milk was not fresh, it proved highly virulent from causes other than that of tuberculosis, and killed the animal within forty-eight hours, indicating the presence of irritant organisms, the presence of which is of serious import, as likely to give rise to the acute and fatal gastro-enteritis of infants which is so exceedingly common during the summer and autumn.

The use of humanised cows' milk, instead of, or in addition to breast milk for young infants, is exceedingly common, and the wide difference between the two must be carefully borne in mind. Milk, when supplied in the natural manner to the young infant, passes direct, without exposure to the air, from the gland of the mother to the stomach of the infant at a suitable temperature and adequately mixed; it contains neither adulterant nor preservative, nor has anything been abstracted from it, whilst, most important of all, it is, bacteriologically, clean and pure. The milk of commerce, on the other hand, is exposed to numberless risks of contamination, not only in the open buckets in the shippon, but also in strainers, pails, milk cans, and jugs, into which it finds its way in the course of its progress to the consumer; and even if it is neither skimmed, watered, nor otherwise adulterated, yet after being hawked from door to door through the dusty streets it must necessarily arrive in a varying degree of staleness, more or less contaminated, and loaded with microbes of various kinds.

Between milk supplied as Nature intended it, and milk which has gone through so many vicissitudes, there is a gulf fixed, and a consideration of these facts indicates the extreme importance of care, and the desirability of sterilisation in all cases of doubt.

Without going into details as to the nature of the putrefying organisms, or how they find their way into the milk, it may be stated that the direct destruction of infant life every summer and autumn from putrefying artificial milk-foods is remarkable. The facts may be illustrated by contrasting the mortality amongst an equal number of infants of equal ages fed respectively upon the breast alone, and upon cows' milk with artificial foods, of which milk, doctored in various ways, is the basis.

As the result of a careful investigation by the writer, extending over several years, and supplemented and checked by the experience of members of the Liverpool Medical Institution, it is found that the mortality amongst equal numbers of infants below three months of age, fed respectively upon cows' milk with artificial foods, as well as breast milk, is fifteen times as great as it is amongst those fed on breast milk alone; or to put it in another way, if twenty infants out of every thousand below three months of age, fed upon the breast alone, die from infantile diarrhœa, no less than 300 die out of every thousand fed upon cows' milk and artificial foods as well as breast milk; but if they get no breast milk at all, nothing, in fact, but cows' milk and other artificial foods, it would appear that the deaths of those under three months of age would increase from 20 to 440 per thousand —a number almost incredible. The enormous

mortality amongst infants during the autumn months is very largely accounted for by this circumstance.

IV. Preservatives and Colouring Matters

Preservatives and Colouring Matters are now very extensively used, and consideration of their probable effects upon the digestive system is necessary.

Preservatives.—The very extensive use of chemical preservatives renders the subject one of considerable importance. It is not necessary to say that all waste in regard to perishable food-stuffs should be avoided as far as possible; the usefulness of the application of cold to this end is thoroughly appreciated, so also is the application of heat in the preservation of tinned foods of various kinds; by these means the food is preserved without the addition of anything likely to be injurious, and, by lessening the price, a good and abundant food-supply is brought within reach of the poorer classes.

But in the case of chemical preservatives another aspect of the question appears, since, whilst the chemical used may check putrefactive changes in the food, it may also check the fermentative processes of digestion.

The preservatives most commonly in use are boracic acid, or borates, salicylic acid, or salicylates, formalin, and of course salt. Boracic acid, or borates, is very largely used; margarine, butter, ham, bacon, sometimes fish, cream, and milk contain it; in margarine and butter it is more or less uniformly mixed, its use has been common for many years, 30 grains to the pound being often met with; in hams and bacon it is usually and chiefly on the outside, although not infrequently from 4 grains to 8 grains per pound are found in the interior. Sausages, pork-pies, and pastry frequently contain it. Salicylic acid and salicylates are found in jam, but the manufacturers appear to be *bona fide* anxious to add only a necessary minimum in this case; what are known as British wines and also certain temperance drinks are frequently found to contain boracic acid or salicylic acid far in excess of what is necessary, the range found being from 4 to 100 grains of boracic acid per gallon, and 7 grains to 150 grains of salicylic acid per gallon. Their use in these larger quantities is distinctly objectionable. It has actually been urged in defence of the practice that the quantities are not larger than those given in medicinal doses; this may be true, but the question is one not of medicine—which may be all very well in its way—but of food, and indiscriminate doses of physic are not wanted at meal-times.

Formalin is not infrequently used to preserve milk and cream. The addition of chemical preservatives of various kinds to milk is commonest during the winter months, probably because at that season the milk has the highest value, and consequently the greatest profit is to be derived from keeping it, so that it may be worth the dealer's while to run the risk of a prosecution.

The pernicious results arising from the indiscriminate use of preservatives have been demonstrated by Professor Boyce, who, feeding three series of five kittens upon milk containing: (*a*) 10 grains of boracic acid to the pint; (*b*) 5 grains of boracic acid to the pint; (*c*) upon pure milk, found that the group fed with 10 grains to the pint became emaciated and died after obvious evidences of gastro-intestinal disturbance, that those treated with 5 grains showed similar results, whilst the remaining five continued perfectly healthy, and showed the normal increase in weight.

Similar experiments with formalin showed, in proportion to the quantity of the formalin used, ill-health and emaciation, whilst control kittens fed with pure milk remained active and well and increased in weight.

Inquiry shows clearly enough that grave danger will follow if young infants are fed upon milk containing these preservatives, and it emphasises the necessity for feeding infants as nature intended them to be fed, or if that is impracticable, to employ only pure, natural, and clean substitutes.

Investigations such as these have also been undertaken by other observers, and show plainly enough that chemical preservatives must be used with extreme caution; their careless and haphazard use is attended with the gravest danger to the consumer.

Colouring Matters.—Sausages, more especially German sausages, are coloured usually with a mixture of borax, and red coal-tar dye of the class known as sulphonated diazol, with a little salt, or saltpetre.

Sometimes ground-rice or bread-crumbs are mixed with the colouring matter.

Armenian bole consists of oxide of iron with a little silicious matter.

A pernicious fraud is perpetrated by brushing-over hams, bacon, fish, etc., with a mixture of borax, salt, red coal-tar dye, and creosote, which gives the article the appearance of having been perfectly and carefully smoked. The name given to the colouring mixture is "Smokene."

With regard to metallic salts, it is relatively uncommon to meet with these; sulphate of copper is found in bottled or tinned preserved peas, beans, etc., sometimes in unnecessary and dangerous quantities; the presence of the salt is injurious and unnecessary, and certainly conceals the inferior quality of the article, for example, by giving to stale and withered peas a spurious appearance of freshness and greenness.

V. The Effects of eating impure Food

The bacillus enteritides sporogenes has been found in unsound meat as well as in unsound fish.

It has already been pointed out that specifically harmful consequences do not necessarily follow from the ingestion of innutritious flesh. Meat from emaciated and worn-out animals is condemned on the sufficient grounds that it is innutritious, and in consequence has not the qualities which the consumer requires. But in regard to the consumption of flesh which is decomposing, or which is taken from animals which have suffered from inflammatory disease, or certain parasitic diseases, the consequences are very different, and few medical men have not from time to time had abundance of evidence to show this; moreover, cooking cannot be relied on to prevent this mischief. Unsound food is liable to give rise to symptoms of gastro-intestinal disturbance, diarrhœa, vomiting, colic, followed by more serious symptoms of septic poisoning, prostration, pyrexia, and failure of the heart's action; many such cases, some resulting fatally, have been recorded. Pies of beef or pork, sausages (alliantiasis) and the like, have also given rise to these conditions. Dr. Ballard (see Reports of the Local Government Board) quotes a number of cases in which mischievous or fatal results have followed the ingestion of animal food; out of fourteen such instances, pig-meat of one kind or another occasioned the illness in no less than nine, veal in one, beef in one, the kind of meat not specified in two, tinned salmon in one. An explanation is suggested of this special liability of pig-meat to produce these specific maladies: of all adult flesh meats ordinarily eaten, pork, under the process of cooking, furnishes the largest proportion of gelatin; young meats, such as veal, are also largely productive of gelatin, and gelatin is a favourite nutriment of morbific bacilli. As a result of his investigation, Dr. Ballard considers that "in infected food capable of producing disease on being eaten, we find one or both of two things—a living microscopic organism and an organic chemical poison of greater or less virulence. Of these two things, that which is immediately operative in the production of the morbid phenomena is the chemical poison which is apparently of a basic nature, and a product of the processes of bacterial life.

"Specifically different bacteria, capable of producing this chemical poison, may through its agency give rise in the human system and in animals to clinical phenomena and pathological changes in the organs which are so similar, that at present they cannot be distinguished.

"Given the bacterium and favourable environment, the bacterium may grow, multiply, and produce its own special chemical poison from the material which affords its nourishment either outside the body or within it."

The presence or absence of an incubation period prior to the manifestation of toxic symptoms is explained by Dr. Ballard as evidence of the symptoms being due either to the operation within the body of the bacterium itself, or of their being due to the operation of the chemical poison already prepared in the food. Where merely the bacterium is introduced, time is required for its growth and for the formation of its poisonous chemical product; when the chemical poison already prepared outside the body is introduced, its operation is more speedy.

Not only is thorough cooking of importance in all cases, but equally so is the observance of absolute cleanliness in every stage of the preparation of the food for the table.

A case of fish-poisoning occurred in Liverpool during last year. Five persons after eating cooked salt fish and pig's cheek, purchased from a stall in a low quarter of the city, became seriously ill with the usual symptoms of food poisoning, and four of the five died the day after partaking of the food, the other one the next day but one. The post-mortem appearances indicated a rapid and intense infection. The bacillus enteritides sporogenes was found in three of the cases. A considerable number of persons who had fed from the same stall at the same time suffered no ill effects. An extended investigation into fish of the kind, both cooked and uncooked, was made, and in various instances the growths, inoculated into guinea-pigs, produced pathogenic action, several of them producing fatal results typical of the bacillus enteritides sporogenes. The clinical features of such cases are referred to further under "Intestine."

It is not necessary to allude further to the possibility of specific infection of, for example, typhoid fever, from contaminated shell-fish, nor the transmission of parasitic disease from infected flesh, to which references have already been made.

Foot. *See* Amputations; Ankle-Joint, Region of Injuries; Deformities (*Club Foot, Flat Foot, etc.*); Food (*Impure, Foot and Mouth Disease*); Foot and Mouth Disease; Milk (*Pathological, Foot and Mouth Disease*); Tabes Dorsalis (*Perforating Ulcer of Foot*); etc.

Foot and Mouth Disease.—The term foot and mouth disease is applied to an eruptive febrile disorder of a markedly contagious nature which affects ungulates, ruminants more especially. The eruption is vesicular, and after the vesicle has burst a very hyperæmic and sensitive corium is exposed. The disease is known under a variety of names, which express the opinions that have been held from time to time of its nature; such names are epizootic aphtha, aphthous fever, eczema epizootien, eczema contagiosa, the vesicular disease, murrain epidemic, etc. It is probable that the disease is an exotic, because, according to the *Annual Report of the Board of Agriculture* for 1898, there is no evidence that the disease ever existed in this country prior to 1839, though the Germans say that foot and mouth disease was

rife in England in the middle of the eighteenth century. Whether such is the case or not, we know from Continental authorities the disease was known abroad during last century, and that in the main it was well described by contemporary authorities. Since 1839 there have been several serious outbreaks of the disease in Great Britain, and during the first two years following 1870, when the Board of Agriculture first obtained returns of the diseases affecting stock, it was shown that 1,149,124 animals had been attacked with foot and mouth disease. In 1894 the disease was eradicated, and the chief veterinary officer estimates that it claimed for its victims from 1839 to its final extinction between ten and eleven millions of head of stock. Since 1894 there have been isolated outbreaks of the disease, the last during February of the present year in Norfolk and Bedfordshire. The disease is always existent upon the Continent, in the Low Countries especially, and it is very probable that the occasional outbreaks we have to contend with may have their origin traced to the commerce with these countries. Fortunately the fatality is not great among animals other than those at the teat; the percentage of fatal cases varies from 1 to 5 per cent; in young animals, however, where the alimentary viscera may show lesions, the percentage may mount as high as 50 per cent or more.

Transmission of the disease to the human subject is of fairly frequent occurrence, especially on the Continent, though less often in this country. The disease may attack only one or two individuals, or may occur in epidemic form. In the course of an epidemic many persons in one locality have been affected, and cases may be observed to terminate fatally. The number of cases recorded in the human subject probably falls far short of representing the actual figures, on account of the difficulty in many instances of making a correct diagnosis. The first case was recorded in 1695. Since then many epidemics have occurred in different countries, and more especially between 1862 and 1869 in this country, though even in 1884 over two hundred persons suffered from the disease in Dover. In Germany, a large number of cases are still recorded each year.

Etiology.—The virus is contained in the saliva, the milk, urine, fæces, and skin secretions, and retains its vitality for months, even for a year. Since the secretions and dejections are infective, it has been observed that the disease spreads along the great trade routes from the east of Europe to the west, and along the railway trunk lines, healthy animals being contaminated by the cattle trucks, stubbles, pastures, and byres that have sheltered the diseased, and by manure, litter, and fodder that have been soiled by the infective secretions. The disease is also spread by the attendants upon the animals, milkers, cattle and sheep dealers, and others brought in contact with healthy stock, after having handled those suffering from foot and mouth disease. It is known that birds suffer from the disease, and these may assist in spreading the disease.

The *bacteria* which have been found in connection with the disease possess merely an historic interest, and none of them represent the specific causal agent, which has not yet been discovered. Numerous bacteria have been found mainly in the vesicles: "Micrococcus aphtharum" (Rivolta and Nosotti), streptococci (Klein), "Streptococcus involutus" (Kurth), and pleomorphic bacteria (Stutzer and Hartleb, van Niessen). The Streptococcus involutus appears to be a normal inhabitant of the mouth of cattle (Sanfelice). A bacillus was obtained by Bussenius and Siegel, chiefly from affected animals, but also from the human subject, in fatal cases. The bacillus somewhat resembles the members of the Coli group, and Siegel has lately admitted that it is not the specific agent. Furtuna and Starcovici, Sauer, and Babes and Proca have also described bacteria in relation to the disease. Bodies resembling protozoa were found by Schottelius, Behla, Piana and Fiorenti, and Jungers, but these bodies are probably not true protozoa, and no etiological significance is to be attached to them. The Report published in 1897-98 of the German Commission which investigated the disease, shows that as a rule no micro-organisms can be detected in fresh vesicles, but if the vesicles are several days old, various bacteria have usually gained entrance into their interior.

The specific virus is chiefly contained in the lymph of the vesicles. The blood serum does not seem to be infective after the local signs of the disease have appeared. Lymph which has been mixed with water and then passed through an unglazed porcelain filter is found to be infective. Hence the filtered lymph must either contain a toxine, which is poisonous in such a degree of dilution ($\frac{1}{7500000000}$) as is hardly credible, or else the supposed micro-organism is so small that it can pass through the pores of the filter. The latter is the more probable explanation, for it has been shown that the disease can be transmitted from one animal to another in succession by means of such filtered lymph. Another point in favour of the virus being a corpuscular substance, is the fact that diluted lymph which has been repeatedly passed through a Kitasato filter loses its virulence (Loeffler and Frosch). Lymph retains its infective power for weeks or months under ordinary conditions, but loses it if exposed to a temperature of 70° C. for half an hour, or to 60° C. for one hour.

Experimental Infection.—Calves and cattle are the animals most susceptible to infection, pigs being less so. The most reliable method of infecting an animal is by the introduction of fresh lymph into the blood-stream. By this

method $\frac{1}{5000}$ c.c. of lymph will cause the disease, but $\frac{1}{50000}$ c.c. fails to cause infection. Infection usually occurs after the introduction of slaver from affected animals into the mouths of healthy ones, but with greater certainty if the mucous membrane be previously scarified. The subsequent disease after such an inoculation is as a rule milder than after natural infection. Infection can take place through the stomach, but it is doubtful whether it occurs after subcutaneous inoculation of lymph.

Immunity may or may not be acquired after natural infection. Thus, an animal after recovering from an attack may be immune for months or even years, or, on the other hand, an animal may suffer repeatedly from the disease. In other cases, again, immunity has been acquired *in utero*. The Commission already referred to investigated a number of important facts in regard to the production of *artificial immunity*. It was found that no immunity was conferred by intravenous inoculation with fresh lymph in doses so small that the disease was not produced, or by subcutaneous inoculation with lymph if the disease were not thereby caused. Nor was immunity acquired with certainty after the use of lymph rendered inactive by heat, nor after inoculation with the blood of animals which had recovered from the disease, and which had themselves been found to be immune. This latter statement confirms the results previously obtained by Schütz and David and Zernecke. Siegel, however, stated that he had obtained a protective serum from animals which presented the specific vesicular eruption, and healthy animals inoculated with this serum were said to resist without any special reaction subsequent inoculation with infective lymph. Hecker also lays claim to having obtained a protective serum from the blood of immunised cattle. The German Commission further found that immunity may be produced by the cutaneous injection of a mixture of foot and mouth disease lymph and vaccine lymph. Calves and pigs are said to acquire immunity by the intravenous injection of a mixture of foot and mouth disease lymph and "immune blood," and the injection of these two substances separately has a similar if less powerful immunising action. In the case of adult animals, one is advised to leave the lymph and the serum in contact with one another for some time before injecting the mixture. Diluted lymph, which has become non-virulent by being repeatedly passed through a Kitasato filter, is likewise said to protect animals without causing the disease.

It is, however, somewhat doubtful whether these statements concerning artificial immunity are really reliable, and at present we are unable to say that there is any method whereby true artificial immunity may be produced. Inoculations are doubtless to some extent protective, but any method aiming at the production of a high standard of immunity tends to cause the disease itself. This fact is well exemplified by the results following inoculation with the "Seraphthin" of Loeffler and Frosch. Seraphthin has neither afforded protection nor rendered milder any subsequent attack, and in some instances it has even appeared to cause an outbreak of the disease against which it was supposed to be protective.

Animals attacked. — Though the domestic ruminants (ox, sheep, goat) are most commonly attacked, the pig is very susceptible, and the horse, dog, cat, and fowls are not exempt. It has also been observed in the camel, llama, giraffe, deer, antelope, buffalo, bison, etc. The disease may be transmitted to man, either by inoculation or by the consumption of milk from diseased cows, or by butter and cheese made from contaminated milk. Cream especially is virulent.

Symptoms.—The symptoms will be described under the two heads of constitutional and local.

Constitutional Symptoms. — The incubative period of the disease varies between twenty-four hours and seven days. Three or four days is common. During invasion the animals show an elevation of temperature, but this need not necessarily be high; in many outbreaks a rise of 2° F. alone has been noticed. The animal isolates itself, stands with the back arched, and has rigors. Saliva may escape from the mouth, and the patient may move stiffly. There may or not be slight abdominal pain, and the same may be said of cough. The respirations are not increased in number usually, though in some cases in the later stages the respiratory movements may be much increased in frequency. The pulse-rate is slightly quickened. If the animal is milking or nursing, the amount of milk furnished by the gland falls off and the quality of the milk is changed: it is of a yellowish white colour and not unlike colostrum in appearance.

Local Symptoms.—The local symptoms in the bovine affect the mouth and the feet. Lesions, however, may be observed upon the skin, mammary gland, vulva, or prepuce of the male. In the sheep, mouth lesions are not common, and in the pig the lesions also are generally confined to the feet, though the snout may also show the eruption.

In the Bovine Mouth.—Prior to the appearance of the vesicles the mucous membrane of the gums and of the lips may appear to be injected, and the amount of saliva present is greater than usual. On the third or fourth day a crop of vesicles put in appearance. These are at first small—no larger than the head of a pin; but they rapidly increase in size by continuing to develop, or by several uniting. The vesicles are found upon the gums, mucous surfaces of the lips, tongue, dorsum, and sides. The vesicles of the dorsum are larger than those upon the sides

of that organ, the hard palate, including the pad covering the toothless premaxillæ. As we have mentioned, the vesicles may become confluent, and individual vesicles may attain the size of a half-crown or more. The fluid contained in the vesicles is at first clear like to water, but later becomes opaque. If the vesicle be ruptured the corium is exposed and found to be intensely hyperæmic. The lesion only involves the superficial parts of the corium, not intruding any great depth into that structure. The epithelium around the wound forms a rounded edge, and soon extends over the denuded corium, restoring the lost covering. Pus is not commonly found upon the exposed tissues, which are constantly flushed by the enormous amount of saliva produced. The epithelium over large areas may be lost; extensive patches from the sides and tip of the tongue, and from the anterior third of the mucous membrane of the hard palate, may be removed. Save in very malignant cases the excoriations are healed in a week or ten days, but a cicatrix is left behind; the papillæ upon the affected parts are not restored. The lesions may extend into the pharynx, and give rise to difficulty in swallowing, or into the trachea and bronchi, and produce cough.

Feet.—The lesions are observed upon the skin in front, just above the hoof (termed by veterinarians the coronet), in the interdigital space, and upon the heels behind. The rudimentary digits may also exhibit the lesion. Vesicles are formed which, on bursting, expose an intensely inflamed corium. Pus frequently is found upon the inflamed surfaces, but usually a scab speedily covers the wound, and healing proceeds rapidly. Sometimes, however, a necrosis sets in and extends deeply, producing an arthritis, a necrosis of the bones, and often, especially in pigs, a loss of hoof. Death results frequently from septicæmia. The foot lesion, which attacks one, two, or even all the feet, induces lameness, and when the animal is standing in a stall the foot is frequently shaken as if to rid it of an offending object. A smacking of the lips, between which saliva is dribbling away, and shaking of a limb in the manner indicated, and lameness when the animal is moved, are almost diagnostic. The presence of the eruption, its character, and several animals manifesting the same symptoms and lesions, make diagnosis certain. The lesions, when observed upon the skin and mucous membrane of the vulva, upon the mammary gland, or upon the prepuce and skin, are similar to those observed upon the feet, and have a similar course.

In Sheep.—The lesions in the sheep are generally confined to the feet, upon the coronet, and most often towards the heels. The animals affected go lame, and linger behind their companions when travelled. The disease may in these animals be taken for "foot-rot," a common disease which is not contagious, but in "foot-rot," which is primarily a disease of the foot, the lesions commence at the toe, and work upwards, whilst in foot and mouth disease the lesions, if the horn-forming structures are involved, extend from above downwards.

In the pig the disease affects the feet most commonly, and in this animal loss of the hoof is not rare. Occasionally lesions are observed upon the snout, and the vesicles may reach a great size.

In the horse the disease is confined to the mouth, but the constitutional and local symptoms in the mouth are similar to those observed in the bovine. Variola and a contagious form of stomatitis may have been taken for foot and mouth disease, but there is at least one authentic case—the subject had licked a cow suffering from the disease.

The disease has also been noticed in the dog and cat. It is also known to attack fowls, vesicles being developed in the mouth and pharynx, upon the conjunctiva, comb, and the membrane between the toes.

In young of all species the disease is serious, and in addition to the local external lesions gastro-intestinal disturbance is indicated, and not infrequently proves fatal. Indeed, the greatest fatality is to be expected among the young at the teat, and milk fed from cows suffering from the disease.

The disease is probably transmitted to man by milk taken from infected cows, or by butter and cheese prepared from such milk, or by direct inoculation from animals suffering from the disease.

The following examples illustrate the methods of transmission: Three veterinarians intentionally drank milk from affected cows; a girl chewed a piece of wood smeared with saliva of a sick animal; again, a wounded hand or finger came in contact with infective buccal secretion; and lastly, an injury was received from the teeth of a sick animal. The symptoms at first are malaise, headache and fever, diarrhœa, and in some cases a rigor and vertigo. About three to five days later, the temperature falls to about normal, and one sees a catarrhal inflammation of the mucous membrane of the mouth and pharynx, accompanied in about one-third of the cases by the formation of small vesicles. These may also appear on the lips, fingers, and toes. The stomatitis in some cases is so severe that small ulcers are formed on the mucous membrane, especially of the gums or the tongue. The urine in most cases remains normal. The duration of the disease is about four weeks. In exceptional cases one finds a skin eruption resembling measles, or vesicles on the skin of the trunk.

The diagnosis in the human subject is often difficult, especially in sporadic cases. The disease may be mistaken for aphthous stomatitis, herpes labialis or pemphigus, and the atypical forms may resemble measles, or even typhoid fever, if the intestinal symptoms be very pronounced.

Post-mortem Appearances.—Few adult animals

die from foot and mouth disease without complications: a septicæmia or a pyæmia may result from secondary infection. Lesions have been noticed, however, in the liver, spleen, and kidneys, changes perhaps induced by the poison of the fever. Ulcerations have been found in the larynx and pharynx, fatty degeneration of the myocardium and pneumonia.

In young animals gastritis and enteritis are not uncommon.

Treatment.—Treatment is simple and resolves itself into (*a*) hygienic, (*b*) dietetic, (*c*) local. The disease, being highly contagious, should be treated as such. The affected should be isolated, and a cordon drawn around the isolation station, and no living thing be allowed to cross the barrier save after rigid disinfection. All litter, refuse, dressings, etc. that have been in contact should be burned, and special suits should be provided for attendants that can be soaked when necessary with a disinfecting solution. The walls and floor of the house in which the diseased have been confined should be flushed down; if necessary scraped, and then washed with solution that will destroy the microbe. Under somewhat similar conditions a one per cent solution of sulphuric acid in water has answered well; crude carbolic acid, sulphate of copper, chloride of lime are useful and cheap. As to the diet, the animals will require cooked nutritious food, soft and sloppy, which in swallowing will produce as little discomfort as possible. Hard food will lacerate the mucous membrane, retard the healing of wounds in the mouth, and may aggravate gastro-intestinal lesions. Local treatment consists in the application of mild astringents and disinfectants to the mouth and to the feet. Mouth lesions are best dressed with borax and glycerine applied by brush; a dilute solution of chloride of zinc in water may be used for application to the feet. If many animals are affected, such as a flock of sheep or a herd of swine, these may be driven through a shallow trough, sunk a few inches into the ground, and containing in it an antiseptic solution. All that is required is that an area, about four yards long by three yards wide, be sunk about six inches below the general level of a paved yard. If the depression be made water-tight by cement, a shallow bath of antiseptic fluid is provided through which the diseased animals may be gently driven.

As to the flesh of foot and mouth patients it is innocuous and marketable, save when profound changes have occurred due to fever. The heads of cattle should be condemned and the feet of all. If the heads of sheep and pigs show lesions, they also should be destroyed. Hides and fleeces should be disinfected: they have undoubtedly in many outbreaks been the vehicles by which the disease has spread.

Footling. *See* Labour, Diagnosis and Mechanism (*Podalic Lies*).

Foramen.—A perforation or opening in a bone, a viscus, or a membrane. *See* Embryology (*Foramina of Monro, etc.*); Heart, Congenital Malformations of (*Patent Foramen Ovale*); Hydrocephalus (*Foramina of Magendie*); etc.

Forceps. *See* Labour, Operations (*Forceps, Craniotomy, Embryotomy*); Teeth (*Tooth Extraction*).

Forcipressure.—The arrest of hæmorrhage by the (prolonged) pressure of spring forceps.

Fordyce's Disease.—Milium of the prolabium or central prominent part of the lip (Hutchinson).

Forearm. *See* Amputations; Arteries, Ligature of (*Forearm*).

Foregut. *See* Embryology (*Embryo of Third Week, etc.*).

Foreign Bodies. *See* Aneurysm (*Treatment, Introduction of foreign bodies into sac*); Appendix Vermiformis, Diseases (*Appendicitis, Etiology*); Bladder, Injuries and Diseases of (*Foreign Bodies*); Ear, External, Diseases of (*Foreign Bodies*); Eyeball, Injuries of (*Foreign Bodies*); Hernia (*Structure, Contents*); Intestines, Surgical Affections (*Blocking of Lumen in Obstruction*); Larynx (*Foreign Bodies*); Nose (*Foreign Bodies*); Œsophagus, Injuries and Diseases (*Foreign Bodies*); Pharynx, Examination of (*Foreign Bodies*); Rectum, Diseases of (*Concretions*); Vagina, Diseases of (*Foreign Bodies*).

Foremilk. *See* Colostrum.

Forest Climate. *See* Lung, Tuberculosis (*Treatment, Climate*).

Forewaters.—The liquor amnii which in labour lies in advance of the presenting part of the child, and is expelled when the membranes rupture. *See* Labour, Physiology of.

Formaldehyde.—A non-official preparation (a gas), commonly called formalin when in aqueous solution (40 per cent), and having the formula CH_2O (formic aldehyde); formalin is a powerful disinfectant and antiseptic, and is much used (weak solution) for hardening microscopical specimens, and (in a stronger solution) for preserving naked-eye specimens; it has been used, but not widely, in surgery and medicine. *See* Disinfection (*Formaldehyde*); Food (*Preservatives*); Lungs, Tuberculosis (*Treatment, Specific Measures*); Mammary Gland, Diseases (*Carcinoma, Palliative Treatment, Formalin*); Milk (*Adulteration, Examination, Detection of Formaldehyde*).

Formalin. *See* Formaldehyde.

Formatol.—A proprietary preparation, containing formaldehyde, and said to act as a disinfectant.

Formic Acid.—Synonym, *Aminic Acid*, H_2CO_3. A colourless liquid, miscible with water in all proportions. *Dose*—2-10 m. The following salts are in common use. 1. *Sodii Formas*, a white crystalline soluble powder. *Dose*—$\frac{1}{6}$-3 grs. *Preparation*—Elixir Sodii Formatis. *Dose*—ʒii. 2. *Potassii Formas*. Properties similar to those of the sodium salt. *Dose*—$\frac{1}{6}$-3 grs. 3. *Lithii Formas*, a soluble crystalline powder. *Dose*—$\frac{1}{6}$-2 grs. 4. *Strychninæ Formas*, a white crystalline powder, soluble 1 in 2 of water and 1 in 6 of alcohol. It combines the actions of strychnine and formic acid. *Dose*—$\frac{1}{50}$ gr.

In strong solution formic acid has a caustic action. It has been injected locally in the treatment of chronic rheumatism, sciatica, etc., 8 m. of a 3 per cent solution being the quantity recommended. It may by preceded by the injection of cocaine to lessen the painful effect. Subcutaneous injections of weaker solutions (1 in 100,000 to 1 in 1000) have been employed for lupus and malignant disease. By the mouth or hypodermically in dilute solution both the acid itself and its salts have a very powerful stimulant effect on striped and non-striped muscle. The contractions are strengthened, the output of work is greatly increased, and the onset of fatigue is retarded. The action is similar to that of caffeine, but more powerful. They improve general nutrition and stimulate the appetite. They have a distinct diuretic action, and in sclerosis of the kidneys the amount of albumen excreted is said to be diminished. The heart is strongly stimulated by a direct action on the cardiac muscle, and if the acid is not given in too concentrated solution there is practically no general arterial constriction. Formic acid and its salts have been prescribed in all forms of want of tone and of muscular debility, in circulatory enfeeblement, in phthisis, in constipation, in over-distension of the bladder, in threatened cardiac failure, and in chorea. The formate of strychnine has been very largely used as a stimulant and general tonic, the additional action of the strychnine being an advantage in some cases.

Formication.—A peculiar sensation, as of ants creeping over the skin, met with in nervous disorders, etc.

Formicin. — Formaldehyde acetamide; an antiseptic.

Formula.—A prescription; also a group of symbols and figures representing the constitution of a chemical compound, or the order and arrangement of a number of structures (*e.g.* dental formula).

Fornix (Pl. **Fornices**).—The roof or vault, *e.g.* of the brain or of the vagina; the latter is generally regarded as divided into four spaces by the protruding vaginal portion of the cervix, and these are named anterior, posterior, and two lateral (right and left), according to position.

Fossa.—A depression, excavation, or wide-mouthed pit, a term frequently used in Anatomy and Embryology.

Fourchette.—Any structure which is arranged in a fork-like manner, but commonly used of the semilunar notch of the sternum and of the posterior commissure of the vulva (union of posterior ends of labia minora). *See* GENERATION, FEMALE ORGANS OF (*Labia Minora*); SYPHILIS (*Acquired*, *Situation of Primary Sore*).

Fourfooted Progression. — A method of treating scoliosis introduced by Klapp; the patient walks on the hands and knees, moving the hand and knee of the same side forward at the same time; the method causes very considerable movement of the spine.

Fourth Disease. (Dukes' Disease, Dukes'-Filatow's Disease.) *See also* RUBELLA; SCARLET FEVER (*Diagnosis*).

In 1900 Dr. Clement Dukes brought forward evidence tending to prove that in addition to the three eruptive fevers ordinarily recognised—scarlatina, measles, and rötheln—a fourth existed. This he called "Fourth Disease," the name by which it is commonly known in this country, while in Germany it is called after its discoverer—Dukesche' Krankheit. As it must be admitted that Dr. Dukes has not established his contention that a "fourth disease" exists to the satisfaction of his critics, it would be best first to follow his description of the malady, and then to enumerate some of the objections which have been taken.

Dukes denies that there are two varieties of rötheln, and asserts instead that two definite and distinct diseases—an attack of one of which does not protect from an attack of the other—have been confused under the same name. The scarlatiniform type of rötheln, for which the name "fourth disease" is now proposed, has the following characteristics:—

It occurs chiefly in spring and summer. The incubation period is from 9 to 21 days. Premonitory symptoms are often absent; in severe attacks there may be headache, anorexia, chilliness, backache, etc. The temperature varies from 98·4° to 103° or 104°, but is not necessarily proportionate to the intensity of the rash. The pulse is accelerated in direct ratio to the pyrexia, but seldom rises over 100. "The eruption is usually the first symptom noticed, and covers the whole body in a very few hours. The hue is a bright rosy red, and is raised somewhat from the surface of the skin, the sensation of heat of the skin to the touch, even when the rash is very full, is much slighter than in scarlet fever." The fauces are swollen and have a velvety appearance, but sore throat is not com-

plained of. The eyes are suffused; there is general pea-like enlargement and tenderness of the superficial glands; the tongue is clean or slightly furred,—never thickly coated; there is comparatively little sensation of illness, and even if severe the symptoms pass off in a few days, and recovery is complete in a fortnight. The rash is followed by desquamation, which may be slight or extensive. A slight rash is usually attended by little desquamation, and what does occur disappears in a week or two. After a full eruption there may also be little peeling, but, on the other hand, it may be as free as in scarlet fever. There are no sequelæ; albuminuria is a rare and transient symptom. Fourth disease is most infectious at the commencement; the infection, never very great, does not endure beyond 14 or 21 days with efficient disinfection, even though slight desquamation be still in progress. No treatment, save rest in bed for a few days, is required.

From the above description it will be seen that the distinction between German measles on the one hand, and mild scarlet fever on the other, is by no means easy. The rash is diffuse, not patchy, as in the former disease, the glandular enlargement is less, and desquamation is much more marked; while, as against scarlet fever, we have the more generalised appearance of the rash at the onset, the comparative absence of pungent heat to the touch, the enlargement of the cervical and occipital glands, the mildness of the sore throat, the absence of the characteristic tongue, and the less frequent pulse. At a later period in scarlet fever albuminuria is common, while desquamation is more prolonged. In a subsequent communication Dr. Dukes also urged, as against his cases being scarlet fever, that in that disease we do not find a full rash without a high temperature and subsequently free desquamation, whereas in fourth disease a copious rash may be unattended by fever and peeling.

In addition to the clinical phenomena, the evidence on which Dr. Dukes relies is furnished by the occurrence of epidemics, chiefly in schools. He has observed concurrent epidemics of fourth disease and scarlet fever, in which some of the patients were attacked by both infections in succession. Moreover, nearly half those affected in one epidemic of fourth disease had already had rötheln previously. The insological position of fourth disease is, according to him, justified by its breeding true, and by its conferring protection against a second attack, but not against scarlet fever or rötheln.

Although Dukes was the first in this country to discriminate this member of the exanthemata, it, or something very like it, had already been recognised by Filatow of Moscow thirteen years earlier, under the name Rubeola Scarlatinosa. He describes it as resembling the mildest type of scarlet fever, and not, as a rule, followed by desquamation. It stands in the same relation to scarlet fever as rötheln does to measles. Filatow was led to identify rubeola scarlatinosa by observing epidemics among children who had already had, or subsequently became affected by, scarlet fever. Filatow offers no proof that in his cases rötheln had previously occurred. From Dresden Unruh has also reported small epidemics of fourth disease; the description he gives tallies in all respects with that of Dukes. Most of his cases had already had scarlet fever and rötheln, and the disease, as he saw it, always bred true. Bokay, of Buda Pesth, is also a strong supporter of the existence, as a separate entity, of fourth disease. Among others who took Dr. Dukes' side are Broadbent, Ashby,—who, however, puts the incubation at 7 to 9 days,—and Weaver, who reports cases among convalescent scarlet-fever patients.

The objections raised to Dr. Dukes' hypothesis fall into two categories: (1) those which are concerned with a detailed criticism of his cases with the view of showing that they were either German measles or scarlet fever, and (2) those of a more general nature. It is impossible within the limits of a short article to go into the first series of objections, but it seems to the writer that very great weight must attach to the opinion of experienced independent observers like Filatow, Dukes, Ashby, Bokay and others, who actually saw the cases, and were satisfied that they were not examples either of scarlet fever or rötheln. One of the more general objections which has been raised is that Dukes places too much faith on one attack of a fever protecting from a second, and that in so far his inferences require qualification. Again, it is urged that if a disease so closely resembling mild scarlet fever existed it would often be notified as scarlet fever, and cause epidemics in fever hospitals, while in their turn the patients would become infected with genuine scarlet fever. As a matter of fact, Dukes and Weaver believe that many so-called relapses of scarlet fever are instances of successive infections with the two diseases. A further series of objections concern the symptoms mentioned above as those which Dukes lays down as distinguishing between scarlet fever and fourth disease. It is contended that many mild cases of the former do not display the symptoms in question. On the whole, the trend of opinion among superintendents of fever hospitals seems to be that a fourth disease does not exist. At present, therefore, the question is still *sub judice*. Probably the solution of the matter will only be arrived at from a study of the epidemiology of so-called fourth disease, since its differentiation from mild scarlet fever by its symptoms alone appears impossible. If this be so, it is to physicians in private practice, and particularly to those in charge of schools and institutions, who have the best opportunities of observing incubation periods, previous protection, and duration of infectivity, that we must look to settle the question.

Erythema Infectiosum.—Under this title Eschenih of Vienna has recently (1904) described another infectious exanthem. Like fourth disease, it has hitherto been confused with rötheln, but, except for this one point of resemblance, the two diseases have nothing in common. So far, no confirmation of the existence of this disease has come from outside Germany, but apparently an epidemic eruptive fever, corresponding to that from which Eschenih's patients suffered, had been described under various names —megalerythema epidemicum, scarlatinoid, morbilloid, erythema simplex margenatum—by a number of observers in that country during the preceding 10 or 15 years. Eschenih, however, was the first to prove to the satisfaction of his countrymen that it was a separate entity, and his view appears to be generally accepted in Germany at the present day. The clinical features of this latest member of the family of the exanthemata are as follows:—It is an epidemic disease, which often follows outbreaks of rötheln, and most commonly affects children between 4 and 12 years. It is feebly contagious, and neither protects from rötheln nor *vice versa*. The incubation is from 6 to 14 days. There are practically no symptoms, except the eruption; pyrexia is absent or slight, there is no implication of the mucosæ, and there are no complications. The rash, however, is characteristic. It appears first on the face, spreading to the body on the second day. The distribution is somewhat peculiar; the face and extremities, particularly the extensor aspects, are most markedly affected, while there is frequently but a very sparse crop of spots on the trunk. The character of the eruption on the face is pathognomonic; the cheeks are swollen and covered with a red blush, giving a turgescent appearance, somewhat suggestive of erysipelas. The margins of the eruption are somewhat sharply defined. This confluent rash is bounded below by the nasolabial folds, and is rendered more apparent by the circumoral pallor, while on the forehead and chin it occurs in more discrete measly papules. On the extremities the rash is morbilliform, arranged in geographical outline, giving the appearance of a lacework, but tending to become confluent about the elbows and buttocks. The duration of the rash is from 6 to 10 days. There is no tendency to cutaneous hæmorrhage, no glandular enlargement, and no desquamation.

Fourth Nerve. *See* Ocular Muscles, Affections of; Brain, Physiology (*Cranial Nerves, Fourth*); Brain, Tumours of (*Localising Symptoms, Cranial Nerves*); Physiology, Nervous System (*Craial Nerves, Trochlearis*).

Fovea.—A small depression or excavation; a dimple; a fossa. *See* Retina and Optic Nerve (*Anatomy, Fovea Centralis*).

Foveola Coccygea. — The little depression or dimple occasionally found over the tip of the coccyx and behind the opening of the anus (called on this account the "post-anal dimple"); it is generally single, but may be double, and it may be associated with spina bifida occulta, with sacro-coccygeal nævus, and with coccygeal fistula or sinus; foveola retro-analis.

Foveola Pharyngea. — A slight depression on the basi-occiput, representing a trace of the median canal of the basi-occiput.

Fowler's Solution.—Liquor Arsenicalis. *See* Arsenic.

Foxglove. *See* Digitalis; Toxicology (*Foxglove*); etc.

Fractures.

See also Ankle-Joint, Region of, Injuries (*Fractures near Joint, of Astragalus, and of*

[1] These exclude fractures in the immediate neighbourhood of joints, for which see "Ankle-Joint," "Elbow-Joint," etc.

Oscalcis); Bone, Diseases of (*Spontaneous Fractures*); Brachial Plexus, Surgical Affections of (*Tears, Causes*); Brain, Surgery of (*Injuries to Brain, in Fractures*); Chest, Injuries of (*Fractures of Ribs, Sternum*); Elbow-Joint, Injuries of (*Fractures*); Hip-Joint, Injuries of (*Fractures of Hip*); Joints, Diseases of (*Neuro-arthropathies*); Knee-Joint, Injuries of (*Fractures in Vicinity of Knee*); Malingering (*Fractures*); Medicine, Forensic (*Wounds and Injuries*); Meninges of the Cerebrum (*Inflammation, Pachymeningitis, Causes*); Mouth, Injuries of the Jaw (*Fractures*); Orbit, Injuries of (*Fractures of Walls*); Pregnancy, Physiology (*Changes in Bone, Delayed Union in Fractures*); Pregnancy, Intra-Uterine Diseases (*Fractures of Fœtus*); Rickets (*Complications, Fractures*); Shoulder, Injuries (*Fractures*); Spine, Surgical Affections (*Fractures*); Trades, Dangerous (*Phosphorus Poisoning and Match-Making, Fractures*); Wrist-Joint, Injuries (*Fractures*).

It is impossible within the compass of this article to do more than bare justice to such an important surgical subject as the fracture of bones. Space, however, can be found to fill in the outline and to draw attention to the advances which improvements in physical methods have rendered possible in diagnosis, and to the changes which Listerian surgery has introduced into their treatment.

Causes.—The causes of fractures are either predisposing or determining. The *predisposing causes* are those abnormal conditions which lead to the atrophy of bone or to alterations in its consistency. Chief amongst them are rickets, syphilis, tubercle, and scurvy; new growths, either primary and sarcomatous, or secondary and carcinomatous, especially when they grow within the bone and weaken it by expansion; hydatids, and such physiological causes as old age, pregnancy, usually about the seventh month, and that interference with the blood-supply to the bone which is often associated with long-continued disuse. Some affections of the central nervous system are also predisposing causes of fracture, the best known being locomotor ataxy, progressive muscular atrophy, disseminated sclerosis, paraplegia, general paralysis, and the congenital defects of the central nervous system associated with hydrocephalus. There is also a constitutional brittleness of bone (osteopsathyrosis or fragilitas ossium) about which we know but little, except that it is sometimes hereditary, and is transmitted strictly in the male or female line.

The *determining cause* of a fracture is always injury, the violence acting either directly upon the bone, as when the ribs are broken by a kick from a horse, or indirectly, when they yield to pressure applied simultaneously to the sternum and the backbone, as often happens in a crowd. The amount of force required to break a bone varies very greatly, and the result sometimes appears to be out of all proportion to the cause, as in cases where a slight slip produces a severe compound fracture, comminuting the bone and lacerating the tissues, whilst in other cases the application of a much greater force may only cause a simple fracture. Such a variation seems to depend in part upon the direction in which the force is applied to the bone. A cylindrical bone resists to perfection a pull in its long axis or a weight applied in its diameter, but it offers very little resistance to a twist and still less to flexion, for there is no doubt that muscular action is able to break even so stout a bone as the humerus.

Indirect violence causes as many fractures as when the force is applied directly to the bone, but its results are very capricious. Thus a fall upon the hand may lead to a Colles' fracture at the wrist, to a fracture of both bones of the forearm, the ulna in the lower third, and the radius at a higher level; to a backward dislocation of the elbow; to a fracture of the humerus in its lower third, or the arm may escape entirely, the clavicle being broken obliquely at the junction of the middle and outer third. A fall upon the feet in like manner may cause a variety of fractures more or less distant from the legs, until it culminates in a fracture of the base of the skull.

Congenital fractures occur from time to time, many, of course, produced during birth, but others—the truly intra-uterine—have taken place during gestation, for they may be found to have repaired themselves before delivery. Some of the intra-uterine fractures are undoubtedly the result of injury to the mother's abdomen, as in intra-uterine fractures of the leg, femur, and clavicle; others again are associated with embryonic malformation, whilst yet others are spontaneous in the sense that no adequate cause can be assigned for their occurrence.

The classification of fractures has undergone recently a very remarkable change. It was all-important for the older surgeons to discriminate between simple fractures, where the skin remained unbroken, and compound fractures, where the ends of the broken bone were exposed either directly or indirectly to the air. A simple fracture healed with them as it does now: a compound fracture always meant prolonged suppuration, and was too often the cause of death to the patient. Aseptic surgery has changed all this, and we now make a fracture compound with as little hesitation as we incise tissues elsewhere. Primary compound fractures, however, are still of grave prognosis, especially when they involve the larger joints, for it is impossible in many cases to render them surgically sterile. Our main classification, therefore, has become anatomical, and a fracture is grouped, first according to its position, and

afterwards as to whether it is complete or incomplete. The complete fractures are further subdivided into single or multiple; simple or comminuted; oblique, transverse, spiral, or longitudinal; impacted or unimpacted; involving the joint or wholly extra-articular, for upon all these points depends the prognosis.

An incomplete fracture is either bent, curved, or indented. It is usually seen in children, the bent or curved variety in the tibia, radius, or ulna of the rickety, and indented fractures in the frontal or parietal bones after falls or the application of midwifery forceps. A green-stick fracture is another form of incomplete fracture, in which the bone is bent so that its convex surface is broken without any necessary laceration of the concave surface. It is often seen in the clavicles and in the bones of the forearm of children, and its name is derived from the resemblance which it shows to the behaviour of a green twig which has been forcibly bent. Except in the clavicle all green-stick fractures should be straightened before they are put into splints. This is especially necessary in the case of the radius and ulna, where great impairment of pronation may result when the bone is allowed to heal in a bent position. In some cases a bone may be broken and its ends may be comminuted without rupture of the periosteum, a condition seen in children suffering from that form of scurvy known as "Barlow's disease." Fissured fractures, such as occur in the skull, the ilium, and the lower jaw, are also instances of incomplete fractures. The difficulties connected with incomplete fractures are rather those of diagnosis than of prognosis or treatment.

Traumatic separation of the epiphyses is only now beginning to receive the attention which the importance of the subject merits. Fractures and separated epiphyses were formerly considered identical injuries, and were treated in the same manner, but surgeons now recognise that the two forms of injury are distinct, that they are attended by separate signs and sequelæ, and that they require different treatment. Traumatic separation of the epiphyses is most common between the ages of eleven and eighteen years, for in young children the epiphyses are so cartilaginous and elastic as to escape injury, whilst later in life they become fused with the shaft of the bone. The separation of the epiphysis is either complete or incomplete. When it is complete the line of separation extends through the epiphyseal line in its whole extent, or it involves a part of the shaft of the bone. Partial detachment or juxta-epiphyseal strain is apt to be overlooked or treated merely as a sprain.

It is often difficult to make an accurate diagnosis when the displacement is slight, but in well-marked cases unusual mobility at the seat of an epiphysis, local pain, with swelling and ecchymosis, afford a clue to the nature of the injury when the age of the patient is taken into account. If crepitus can be obtained it differs from that occurring in fracture because it is "muffled," that is to say, it lacks the crispness of bone moving upon bone, for it is caused by cartilage rubbing against the uneven end of the diaphysis. The delicate tissues of children lend themselves especially to the use of the X-rays, either by screen or plate, in the elucidation of these injuries.

The prognosis depends partly upon the nature of the injury and partly upon the treatment. Simple separations in healthy persons heal as readily and with less deformity than simple fractures, but in unhealthy persons abscess, periostitis, and tubercular disease are not uncommon. The especial dangers to be feared are paralysis and gangrene due to pressure of the displaced epiphysis; the more remote dangers are permanent deformity, impaired movement in the neighbouring joints, and either partial or complete arrest of growth in the limb. Infective osteomyelitis is sequel of compound separation of an epiphysis, though it may also occur in the simple forms.

The treatment of a separated epiphysis consists in immediate replacement in the compound as well as the simple forms of injury. When the displacement is considerable an anæsthetic must be administered, and reduction should be brought about as methodically as in a case of dislocation. The injured portion of the limb is then encased in some form of moulded splint, and the circulation through the part is carefully watched for a day or two. Massage is applied earlier than in fractures, and in a simple case may be commenced on the tenth day.

Every complete fracture is associated with a certain amount of displacement of the two ends of the broken bone, the amount depending upon the position of the injury and the condition of the ends of the bone. It is either sliding or angular, simple or twisted, in unimpacted fractures, and it is part of the art of surgery to render the displacement as slight as possible, and to keep the ends of the bone in the most accurate apposition until the injured part is able to repair itself.

Diagnosis.—The diagnosis of fractures has greatly improved since the use of the Röntgen rays has enabled the surgeon to view the fragments on the screen and to obtain skiagraphs of the broken bones, because many injuries which were formerly recognised with the greatest difficulty are now rendered perfectly clear. In large towns, in public institutions, and in private practice, it is advisable to skiagraph every case of fracture a few days after the bone has been set, in order to verify the accuracy of the position. The negative can be obtained without moving the limb and in ordinary daylight, but it is necessary to use a wooden or plaster splint.

The diagnosis of a fracture depends upon signs which can be verified by the surgeon, and symptoms which require the descriptive powers of the patient for their elucidation. The diagnosis of a fracture is often easy, but there are many cases in which it can only be made with the very greatest difficulty, and there are some in which the nature of the injury remains for ever undetermined, or is ascertained so late that the surgeon is held blameworthy.

Methods of Examination.—A routine method of examination should therefore be adopted in every case in which there is a suspicion of fracture, and although the symptoms are less trustworthy guides than the signs, they should be determined first by questioning the patient, who will thus become accustomed to the surgeon's examination. Pain and loss of power in the limb are the two chief symptoms of fracture of a long bone, although both may be fallacious. Pain is an important symptom if it can be shown to attain a maximum over a given spot directly after an injury, and if the same point of maximum pain is indicated when the bone is fixed above and pressure is made upon the shaft at some distance below the seat of injury. But the sudden onset of localised pain is a characteristic feature of some forms of acute inflammation of bone. On the other hand, absence of pain in a case of undoubted fracture raises a suspicion of chronic alcoholism (when a surgeon is on his guard for the appearance of delirium tremens or traumatic delirium), or it points to locomotor ataxy or other disease of the central nervous system. The loss of power over the limb is often a well-marked symptom of fracture, but it is absent in cases of impacted fracture, or when only one of a pair of bones is broken in the arm or leg. A simple contusion, however, especially over the hip, often causes great loss of function, though the impairment is only temporary.

The signs of fracture are much more important than the symptoms. They should be elicited methodically, taking care to give the patient the least possible pain. The examination should be made at the earliest opportunity before the swelling has obscured the landmarks, and the least painful part of the examination should be undertaken first. In every case the injured part should be exposed completely, that the surgeon may examine it thoroughly, and it should be compared carefully with the corresponding part upon the opposite side. The examination should be continued until the surgeon has decided upon the diagnosis, for diagnosis in fractures is the key to successful treatment. In every case, too, care must be taken not to overlook other injuries when a fracture has been detected, especially in cases near a joint, where a dislocation may complicate the fracture. And even though a fracture is simple when it is first seen, the ends of the bone and the skin should be examined to ascertain whether it is likely to become compound.

Every fracture is associated with some deformity of the broken bone, although the deformity may be masked by the thickness of the overlying tissues, as in the hip, or by the support obtained from neighbouring bones, as in the ribs, the ulna, the radius, and the fibula. In fissured fractures the deformity is so slight that it may pass unnoticed.

Most fractures are associated with some alteration in the length of the injured bones, which become shortened, but the alteration may be so slight as to fall within the limits of error or of natural development. Every fracture of a long bone, therefore, must be compared carefully with the sound side by means of a tape measure, taking care to measure from similar bony points on the two sides.

Abnormal mobility in the shaft of a bone is a certain sign of fracture, but it is wanting in greenstick, curved, and fissured fractures as well as in the great group of impacted fractures.

Crepitus, or the sensation produced when the two ends of a recently broken bone rub against each other, is diagnostic of a fracture; but it may be mistaken by the inexperienced for the creaking of a tendon moving in an inflamed sheath, as in tenosynovitis; for the rustling of melon-seed bodies in a bursa or ganglion; for the slight stickiness which accompanies effusion into joints; for collections of blood containing blood-clots; and for the crackling of emphysema. There is no crepitus when the ends of the bone are widely separated, as in fractured patella, when there is impaction, or when a mass of tissue intervenes between the two fragments. Although crepitus, felt by an experienced person, is a certain sign of fracture, it is always undesirable to obtain it by rubbing the ends of the bone together. The aggregate of the other signs is usually sufficient to determine the nature of the injury, and the production of crepitus is very painful to the patient; besides, it may do harm to the surrounding tissues and even to the ends of the bones themselves.

An extensive ecchymosis, first seen some days after the injury, and the appearance at a still later date of the lump due to callus, are valuable aids to diagnosis in some very obscure cases, as in fractures about the hip and shoulder, and in greenstick fractures.

Process of Repair.—The pathology of the repair of fractures is difficult, because the details have not yet been completely investigated. The method differs somewhat in aseptic and in septic fractures. Aseptic or simple fractures are repaired by callus, a material produced by the fibrous tissues in the immediate neighbourhood of bone, and therefore derived from the bone itself, from periosteum, and from the surrounding connective tissues. The injury which produces the fracture tears all the soft

parts so that the blood is poured out in considerable quantity both from the periosteum and from the vascular medulla. The blood remains fluid for six or eight days, by which time the periosteum near the fracture is swollen, and its fibres are dissociated by exudation and the proliferation of cells in its deeper layers. This forms the first or hæmorrhagic period in the repair of a fracture; it is of very short duration in such vascular bones as those of the face, which heal almost by first intention, and it is proportionately prolonged where severe injury has given rise to much laceration, or where disease, as in scurvy and hæmophilia, has led to a great effusion of blood. The second, embryonic or cartilaginous, stage is that in which inflammatory tissue is produced in abundance on every side by the periosteum, the Haversian canals, and the medulla. In adults the inflammatory cells are abundant and the cartilage cells are few, but in animals and in children the cartilage cells may be very numerous. The blood is absorbed and the embryonic tissue or blastema becomes converted into fibrous tissue, the organised callus. The portion of callus lying farthest away from the bone becomes organised more rapidly than that which lies between the broken ends and within the medullary canal. New bony tissue is then formed beneath the periosteum and within the medullary canal by the ordinary process of intra-cartilaginous or intra-membranous ossification, and this constitutes the third stage in the repair—that of provisional callus—whose formation and consolidation lasts on an average from fifteen to forty days.

The provisional callus is at first porous, but the final process of repair is one of modelling, which lasts for months or years. The modelling process is a combination of a rarefying osteitis at the periphery, combined with sclerosing osteitis at its centre, and it continues until the callus is converted into normal bone, and until all sharp edges, spicules, and fragments of the original bone have been rounded off. It must not be thought, however, that these stages exist independently of each other or have well-defined limits. They are all processes in the general train of repair, and so pass insensibly into each other.

Repair in a septic or compound fracture takes place in a manner similar to that just described, but the process is masked, hampered, and modified by the suppuration. The callus is less uniformly deposited, granulations are formed in abundance, and the bony fragments are joined ultimately by the union of granulations, which anastomose one with another, and rapidly become converted into bone.

Many pitfalls surround the repair of fractures. The process may be completed so quickly that the bone is firmly united in a bad position before the surgeon has set it, or even has realised that he is dealing with a fracture. This is most likely to happen in a broken nose or in a greenstick fracture. Repair may be unduly delayed on the other hand, or it may never take place at all, as is most frequent in the humerus, in the tibia, and in the femur, where it is difficult to keep the two fragments motionless. In other cases so much callus may be produced as to lead to serious difficulties by the pressure which it exercises upon the nerves or blood-vessels in the neighbourhood.

The Prognosis of a primary fracture involves two separate considerations, the danger to life and the danger to the limb. The danger to life depends in part upon the general condition of the patient, his age, and his freedom from renal and bronchial troubles; and in part upon the character of the individual fracture, which may lead to injury of the brain, lungs, or pelvic viscera. The prognosis in regard to the utility of the limb after a fracture must always be guarded, even in the most simple forms of injury, because, although the result depends to a great extent upon the vigilance of the surgeon and his attention to detail, there is an element of uncertainty which renders it most unwise to be too sanguine either as to the duration of treatment or the usefulness of the limb. Extensive series of examinations of fracture by means of the Röntgen rays have shown that perfect union is rare, and that oblique fractures generally have some overlapping of the broken ends, whilst in deep-seated bones there is often much bony deformity which it is impossible to recognise by the ordinary diagnostic methods. The prognosis becomes more grave, therefore, when the bone is much comminuted, when the line of fracture is very oblique, when it is close to a joint, and when the large vessels and nerves of the limb are injured.

It will be the duty of the surgeon in some cases to recommend amputation of the limb or excision of a joint in some cases of fracture. The operation is either primary or secondary. A primary amputation is required when the limb is hopelessly shattered, as in railway smashes, and in the extensive comminuted fractures caused by explosions, or the passage over a limb of such heavy and slowly-moving objects as drays and tram-cars. The duty of the surgeon is quite plain in these accidents, but there exist a great group of doubtful cases which often exercise the highest art of the surgeon to decide whether he should amputate at once or should wait. On the one hand he may sacrifice a limb unnecessarily, on the other he may cause the death of his patient by delay. Young and healthy adults may recover after excessive laceration of the soft parts with extensive comminution and denudation of the bone, after implication of a large joint, after the main vessel has been ligatured, and the main

nerve sutured. In children and boys I have a great distrust of compound separations of the epiphyses, 'especially when they occur at the shoulder and knee, for osteomyelitis readily occurs, and the patient dies even when amputation has been performed. Old people, I think, should undergo amputation in doubtful cases, whilst children and adults should receive the benefit of the doubt. Secondary amputations are often more hazardous than primary operations, because the condition of the patient is less satisfactory. A secondary amputation is required in gangrene, where there is septic absorption associated with osteomyelitis or extensive suppuration, and as a last resource in cases of non-union or union in such a bad position as to render the limb useless.

Treatment.—Success in the treatment of a fracture depends upon the maintenance of the broken ends of the bone in the best apposition possible until they are soundly healed, taking care that the surrounding tissues are neither crippled by disuse nor are involved unduly in the accompanying inflammatory changes. The objects to be attained by treatment in every fracture of a long bone are firm, bony union, good position, and a useful limb. The treatment of fractures resolves itself, therefore, into reduction with subsequent fixation.

Reduction is the manipulation employed to bring the broken ends of the bone into their natural relation to each other. It may be necessary, useful, or harmful; easy, difficult, or impossible. Reduction without any delay is necessary in a depressed or punctured fracture in the skull, since cerebral symptoms may be produced; in a broken nose, because the nasal bones unite so rapidly that the deformity very soon becomes permanent; and when the sharpness of the fragments threatens to convert a simple into a compound fracture. Reduction is useful in nearly every simple fracture of a long bone, but it may be positively harmful in some cases of impacted fracture, as in those connected with intra-capsular fractures of the hip and shoulder, or in a fracture of the outer table of the skull. Reduction is easy in most fractures, but impaction, muscular spasm, or the interposition of the soft tissues, may render it difficult or even impossible to bring the two ends of the broken bone into good apposition; although early reduction is desirable in most fractures. No attempt should be made, therefore, to reduce a fracture until the surgeon is ready to "put it up" permanently, and until everything is ready the injured part should be kept at rest by a temporary splint. A bandage and sling may be sufficient for the arm, but in the case of the leg the limb must be supported by improvised splints, by securing it to a pillow with a bandage placed above and below the seat of fracture, by sand-bags, or by the help of an assistant to restrain the awkward and involuntary movements which may so easily convert a simple into a compound fracture, or may even drive a sharp fragment of bone through a large artery or vein.

Such care is especially needful when the patient has to be moved from the place of accident to his house or to the hospital, since helpers are more often willing than handy.

Reduction is effected by extension and counter-extension, the force being applied whenever it is possible by assistants, that the surgeon may have his hands free to manipulate the injured part and to apply the splint as soon as he has satisfied himself that the position of the broken ends is sufficiently satisfactory. There are many cases in which mere force will avail nothing, and the surgeon must resort to a variety of manœuvres before he can reduce the fracture, and allusion will be made to this in dealing with the fractures of individual bones. Anæsthesia is often a valuable aid when reduction is difficult, but the surgeon who has to treat such a case in private must bear in mind that the patient's struggles whilst he is becoming unconscious, or unskilled efforts to restrain them, may lead to serious injury at the seat of fracture.

The broken limb must be fixed securely as soon as it has been "set," for it is characteristic of fractures that the deformity is reproduced as readily as it is reduced. It is usually impossible to obtain perfect apposition, for the deformity is produced by the whole of the soft parts surrounding the bone, and not by the action of the muscles alone as was formerly thought; still the deformity can often be diminished by a skilful surgeon.

Fixation is of the greatest service in cases of fracture. It lulls the pain, allays or prevents inflammation, and favours repair with the least possible amount of callus. But the method of fixation has been carried to excess, for it has been used to the exclusion of all other methods of treatment. It has, too, the disadvantage of causing atrophy of the bones, muscles, skin, and connective tissue, whilst the joints and tendons may become hampered from prolonged disuse. It is not surprising, therefore, that a very justifiable reaction against the employment of fixation has taken place during the last few years. The reaction has been carried to an extreme by some surgeons, but there can be no doubt that the early release of a limb from splints, passive movements, skilfully applied shampooing, and the wiring of certain fractures, has been attended with much better results than the older practice of complete fixation carried out rigorously for many weeks. Every case must be treated on its merits, and not on a principle of routine. Success depends very largely upon the vigilance of the surgeon, and upon the attention which he gives to details apparently trivial and minute.

Splints and plaster of Paris bandages or cases form at present the most usual and easiest

methods of securing physiological rest for a fracture.

The variety of splints is infinite, and it passes the ingenuity of man to number them. It may be taken for granted that the simpler the splint the more useful it will be, and that a surgeon with the help of a carpenter and a blacksmith should be able to manufacture all that he is likely to want in his everyday practice. Wooden, plaster of Paris, and poro-plastic splints are better than metal, for they do not interfere with the passage of the Röntgen rays; and whenever it is possible a skiagraph of the limb should be taken after the bones have been duly set and secured in a splint. Wooden splints are made of white pine, the width selected being always a little greater than that of the limb, so that injurious pressure may be avoided. The splint must be well and firmly padded with tow, the fibres being drawn roughly parallel to each other and sewn into a cover of old linen. The skin must be well washed with soap and water, gently dried and dusted over with oxide of zinc or starch powder, and all bony prominences should be protected by padding round them, not over them, with absorbent wool. In no case must skin be allowed to touch skin if the two surfaces are to be kept at rest, and a little wadding or a layer of lint must be placed between them. A bandage must never be put round the limb beneath a splint, and as a rule no bandage should be applied over the seat of fracture. The splint is attached to the limb by strips of plaster, which must encircle the limb spirally and never circularly. It is a cardinal rule that if a splint is uncomfortable it must be adjusted and readjusted until the patient no longer complains, though at first it is always irksome. The limb must be carefully watched after the application of a splint to see whether it swells. Œdema is a sign that there is some interference with the circulation, and a defect in the application of a splint is the most common cause of swelling after a fracture.

Massage combined with fixation appears to give better results in many cases of fracture than the use of splints alone. Some surgeons trust to massage and rest, discarding the use of splints, but this I believe to be dangerous, and I have obtained most satisfactory cures by following the method adopted by Mr. W. H. Bennett, who thus describes the technique (*The Lancet*, vol. i. 1898, p. 359): "The treatment is very simple, and is easily acquired by any person of ordinary intelligence possessing a light hand and a fair sense of touch, gentleness being the keynote to successful manipulations. The method comprises three stages: (i.) Gentle rubbing in an upward direction over the fracture with a view to soothing the patient, the relief of muscular spasm, and the rapid absorption of extravasated blood, etc.; (ii.) Passive movements of the joints above and below the fracture (thus effecting 'internal massage'), by which all matting of the soft parts at the seat of fracture and about the joints is prevented; (iii.) The development of wasted muscles by the ordinary massage processes. The details will be best understood by describing an ordinary straightforward case of fracture of both bones of the leg three or four inches above the ankle, in which there is little or no difficulty in keeping the bones in fair position. Reduction of any displacement of the fragments having been accomplished, the limb is placed upon a back splint reaching above the knee, with a footpiece to which the foot is fixed by a bandage in the ordinary way, care being taken to include no more of the leg above the ankle than is absolutely necessary; a second bandage or piece of wadding fixes the limb to the splint just below or at the knee. As much as possible of the area of the fracture should be left exposed. Rubbing by a gentle, smoothing movement upwards from the ankle is now made by the flat of the hand, grasping as much of the circumference of the limb as is possible. However tender the parts may seem at first, no pain will be caused, but on the contrary a soothing effect is rapidly produced. Ten minutes of this rubbing is sufficient at the first application. If at the end of this time the patient is fairly comfortable, the toes are taken altogether between the operator's thumb and fingers, and very gently extended upon the metatarsal bones two or three times. At the end of the 'sitting' side splints or sand-bags are used in addition to the back splint for the better steadying of the fracture. This proceeding is repeated daily, or oftener if practicable, for a period of four to seven days, the time occupied by each massage being gradually increased to twenty minutes or more—the side splints being removed before the commencement of each rubbing and afterwards replaced. At the end of this time, if the fracture is in good condition and the fragments show no sign of altering their position, the bandages are removed from the foot and ankle, leaving the limb exposed and lying upon the splint. The smooth rubbing already described is now applied over the foot, ankle, and leg for about ten minutes, and then, without removing the leg from the splint, the operator gently flexes the ankle two or three times or more on the leg with one hand whilst he steadies the fracture with the other, the bandages being afterwards replaced as before. This is repeated daily for three or four days, after which the limb at each sitting is gently lifted off the splint on to a flat pillow; the rubbing is now more thoroughly done, and the passive movements of the ankle more freely carried out, the fracture being still, of course, supported with one hand of the operator; at the end of each sitting passive movement of the knee is now added. The passive movement of

the ankle must be commenced very gently, as some slight pain may be caused by 'the internal massage' resulting from the working of the tendons and muscles in immediate relation with the fracture itself. At the end of another week the union is generally sufficiently firm to allow of all the manipulations of ordinary massage, and the patient may be encouraged to move the ankle spontaneously as freely as possible, the fracture being fixed with some form of short splint. The complete massage should be continued until the union has firmly consolidated; the period necessarily varies in different cases, but in a simple, uncomplicated case of fracture of both bones of the leg a month is the approximate time. For the first fortnight the patient is better confined to bed; after that he may lie on the sofa, and generally be allowed to get about on crutches, in which case a moulded poro-plastic or leather splint, made so that it is easily removable for the massage sittings, may be desirable." In the early part of the treatment of fracture by massage it is essential to success that the broken ends of the bone should be kept absolutely at rest, and unless the surgeon shampoos the part himself, or entrusts it to an experienced person in whom he has implicit confidence, it is better not to adopt the method at all, but to leave the massage until the ends have united. Massage is not necessarily contra-indicated in compound fractures. Especial care must be taken in such cases to prevent the infection of the wound. Shampooing is unsuitable when the skin is extensively injured, when there has been so great an effusion of blood just beneath it as to form a large hæmatoma, and where from the seat of the injury it is difficult to keep the fragments in apposition, as in fractures in the upper third of the humerus and the lower third of the femur.

Ambulatory.—The judicious application of various forms of splint, plaster case, or plaster bandage, is often of service in allowing a patient with a broken leg to go about his business before the fracture is firmly united, and this is particularly useful in a fractured fibula, or when a portion of the internal malleolus of the tibia has been torn off. Such ambulatory treatment of a fracture requires that the patient should be kept strictly under observation, as the dependent position of the limb or the imprudence of the patient may easily cause a disaster. The foot and leg must be separated from the plaster of Paris case by a padding of cotton wool at least an inch thick, and this padding must be further increased at the heel, so that in walking the patient puts his heel to the ground before the toes on the injured side.

Wiring.—Even simple fractures are now often treated by cutting down upon the ends of the bone, wiring, pegging, or screwing them together, and afterwards closing the wound. This treatment is most frequently used in transverse fractures of the patella, where a tedious and incomplete convalescence extending over many months is thus replaced by a rapid recovery within a few weeks. Every patient with a broken knee-cap must not be treated in this manner, nor every fracture. A healthy person in the prime of life with a fracture across the middle of the patella gives the best results, but even then the surgeon must be certain of his own ability to carry his methods to an aseptic issue, for an operation which fails often costs the patient his limb and may seriously endanger his life. But the operation of wiring is by no means confined to fractures of the patella. It is often serviceable in very oblique ununited and badly-united fractures, though so far as my experience goes it is most unsatisfactory in the treatment of ununited fractures occurring in children. Simple or aseptic fractures may be wired when injuries to the nerves or blood-vessels require an incision at the seat of injury, whilst many cases of compound fractures likely to become septic can be wired at the time the wound is enlarged to disinfect the injured parts.

Sequelæ.—The consequences or sequelæ of fractures are many. *Stiffness* is the commonest, due either to the existence of adhesions between the muscular and tendinous sheaths and the injured bone, to degeneration and contraction of the muscles, or to the formation of adhesions in a joint which is not perfectly healthy, or which has been injured at the time of the fracture. Massage or the hot air treatment is often of the greatest service in these cases. If the hot air treatment be adopted the limb should be well covered with flannel, and the moisture should be reduced to a minimum by occasionally ventilating the apparatus. Each sitting, after the first, should last an hour, with the temperature beginning at 300° F. and rising rapidly to 380°-400° F. The impairment of movement may be caused by the overlapping of the united ends of the broken bone, by venous troubles associated with injury to the veins, or to thrombosis starting in the medullary veins. These, however, are comparatively trivial consequences of fracture, and appropriate treatment will usually cure them in a longer or shorter time.

Delayed union, fibrous union, and non-union are more serious, but fortunately much less common sequelæ. An ununited fracture is sometimes the result of such constitutional causes as scurvy and chronic nephritis, which weaken the patient, but it is much more often the result of causes acting locally. Any mechanical impediment to the apposition of the ends of the bone may lead to non-union, and of these the intervention of a piece of torn muscle, part of the aponeurosis, or the synovial membrane of a joint, are the most common. But want of rest is the most frequent cause of an

united fracture, for delayed union or non-union is least often seen in the practice of those who are most careful to keep the ends of the bone quiet and in apposition. Slight rotatory movements of the ends of the bone upon each other do not seem to influence the process of repair, but hinge movements may entirely prevent it. For this reason care must be exercised in treating children's fractures by plaster of Paris bandages, especially in cases of broken leg. Their limbs soon shrink from disuse, and a plaster case which fitted admirably at first may become too large in the course of a week, free movement being thus allowed between the ends of the bone. The result is most disastrous, the bones atrophy, all attempts to cause union fail, and the limb becomes so useless as to require amputation. The prognosis in adults is better, for delayed union may often be remedied by putting up the fracture more securely and for a longer period, whilst non-union may be treated successfully by wiring or otherwise keeping the two ends of the bone in accurate apposition.

Complications.—The complications of fracture are very numerous, and it is only possible here to allude to some of the most common and important. Amongst the general complications are shock, exhaustion, collapse from hæmorrhage, delirium, œdema of the lungs, and bedsores. Amongst the local complications of fracture are the various conditions which tend to convert a simple into a compound fracture, and foremost amongst these are sharp ends and excessive bruising of the skin, with so much extravasation of blood into the subcutaneous tissues as leads to sloughing. A large *extravasation of blood* may therefore be laid open with advantage, the blood being washed out and the skin afterwards brought together with interrupted sutures. Other local complications which require the attention of the surgeon are coexisting dislocations and injuries to nerves, arteries, and veins at the seat of fracture. Osteomyelitis, necrosis, pyæmia, and infective or spreading gangrene are more especially complications of compound fractures. *Gangrene* may occur from a variety of causes, constitutional as well as local. It may be infective from the presence of a micro-organism, in which case it is usually fatal, or it may be associated with diabetes or other disease leading to general debility. On the other hand, mortification after a fracture may be purely mechanical in its origin. It is then associated with severe bruising, with acute inflammation in badly-nourished tissues, or to pressure of bandages unduly exercised. Care must be taken, however, in these cases to eliminate other causes before the surgeon commits himself to an opinion that the gangrene has been produced by the pressure of splints or bandages applied by another person. I well remember the case of a man who had broken his ulna, in which the house surgeon was blamed for allowing such a catastrophe to occur. Subsequent dissection showed that the radial artery had been tied years before on account of a punctured wound, and that the ulnar had been pressed upon by the broken end of the bone until the blood-supply had become insufficient for the needs of the part.

Some *clotting of the blood* in the torn veins and medullary sinuses takes place after every fracture, and if the thrombosis is extensive it is an important factor in the œdema which is so troublesome a complication of many fractures. Portions of the clot may be carried away and may lodge as emboli in the smaller branches of the pulmonary and cerebral vessels. These emboli have been observed at various times after the fracture, but they are most common between the twenty-second and thirty-seventh day after the injury. When the medulla of the bone has been extensively crushed, or when it has been softened by inflammation, portions of liquefied fat may be carried to the lungs, brain, and spinal cord, where they block the capillaries and form "fat emboli." These *fat emboli* are not to be mistaken for the post-mortem clots containing fat globules which are sometimes seen in the bodies of persons who have had diabetes. The true fat emboli are generally found where there has been much crushing of the bone associated with large openings in the veins, and so with extensive extravasations of blood. They are produced earlier than the ordinary embolus from thrombosis, so that the symptoms are generally observed within a few days of the fracture. Their presence is marked by transient attacks of dyspnœa, irregular action of the heart, slight hæmoptysis, and Cheyne-Stokes breathing. Collapse with marked pallor of the skin and mucous membrane soon ensues, with spasms of various kinds, or bilateral paralyses and a diminution of reflex irritability. The chest is free from dulness or râles. The temperature varies so much in the different cases recorded that it is useless as a guide. No secondary abscesses are formed, but fat has been detected occasionally in the urine. Packard thinks that such a condition should be treated by the intravenous injection of ether and by the administration of diffusible stimulants.

Muscular spasms occurring in the injured limb are often a very painful complication of fracture. They appear about the sixth or seventh day after the injury, and are especially troublesome at night. They are most often caused by some slight error in setting or fixing the broken bones, and they can often be cured by taking the injured limb off the splint and carefully readjusting it. If they persist in spite of local treatment a suspicion of chronic alcoholism, or of an unstable nervous system, may cross the mind of the surgeon. In very exceptional cases muscular spasms herald the coming of tetanus.

Constitutional Symptoms.—First in importance amongst the constitutional consequences of fractures is that condition, often associated with old age, in which confinement to bed leads to the group of symptoms known as "hypostatic congestion." Inability to sleep, a little cough, slight wandering of the mind, associated with a progressive rise of temperature, show that the patient's heart is too weak to perform its functions, that the tone of the vaso-motor system is impaired, and that the lungs, having lost much of their elasticity, are little more than fibrous bags. Confinement to bed in such cases leads to œdema of the lungs and a pneumonic condition which soon ends in death. The patient must therefore be propped up in bed, or placed upon a couch, to secure the most favourable condition for his pulmonary circulation, but the surgeon need not abandon hope of obtaining good bony union, for he will make use of some of the many forms of plaster of Paris appliance.

Delirium is not an uncommon accompaniment of fracture. Traumatic delirium coming on within a few days of the injury is seen both in children and in old people. It is sometimes met with in overwrought people who have traded too much upon their reserve of nerve force. The delirium is of a low, muttering variety without any rise of temperature, and it has but little to distinguish it, except the cause, from delirium tremens. The prognosis is bad in old people, good in children and the middle aged, if it shows signs of yielding to the ordinary remedies of rest, sedatives, and diet. Delirium tremens is still far too common after fractures in every class of society. The onset is often very rapid. The delirium is active, and may be without any rise of temperature, though there is nearly always a temporary albuminuria. The patient shows a remarkable insensibility to pain, which is most disastrous to his perfect recovery, for he often succeeds in making a simple fracture compound, and he always prevents any accurate apposition of the ends of the bones in spite of every precaution the surgeon can adopt. The principles of treatment are to evacuate the contents of the stomach and large intestine, to restore the appetite, to procure sleep, and to prevent syncope. There is a third form of delirium associated with septic absorption, and associated, therefore, with a rise of temperature. This delirium is worse at night than in the daytime, and it ends when the suppuration is fully established.

A broken bone often remains a source of trouble after it has healed. The callus may be so exuberant as to lead to pressure or impairment of movement; it may be painful, especially in rheumatic, gouty, syphilitic, and agueish persons, or the pain may be due to direct inclusion of a nerve. Callus has been known occasionally to disappear altogether, so that a fracture which seemed to be firmly united has become ununited. It is not very uncommon for a sarcoma to appear at the seat of a fracture; bony and cartilaginous tumours have also been observed.

Fracture of the Clavicle.—A broken collar-bone is perhaps the most common fracture in childhood and youth. The frequency with which it occurs diminishes after the age of twenty, and it is a rare accident in persons over fifty years of age. Fracture of the shaft of the bone is more often the result of indirect than of direct violence, so that it is most commonly produced by falls upon the shoulder, upon the elbow, or upon the outstretched hand. Direct violence causes a fracture at the part struck, and though there are well-authenticated instances of a healthy collar-bone being broken by muscular violence, this accident is most often seen in bones weakened by such chronic inflammatory conditions as scurvy or syphilitic periostitis. The fracture is rarely compound, multiple fracture is not very uncommon, and both bones may be broken.

Immediately after the accident, in the case of simple fracture of one clavicle, the patient nurses his elbow with the opposite hand, and inclines his neck towards the injured side to relax the trapezius and sterno-mastoid muscles. This attitude is not absolutely characteristic of a broken collar-bone, for it is assumed in fractures of the scapula and in other injuries of the shoulder, but it serves to direct attention to the clavicle. The pain varies greatly in amount. It is not severe in a transverse fracture with only slight displacement, but in a very oblique fracture with much displacement the pain is great, and is caused partly by the sternal fragment pressing against the skin, and partly by the pressure of the outer end upon the large nerve trunks which pass below the clavicle.

There is usually no difficulty in making a diagnosis of the oblique fracture at the junction of the outer with the middle third, which is the ordinary result of indirect violence. The affected shoulder is narrower and more sloping than its fellow, whilst the bone lies so superficially that it is easy to feel any inequality. The displacement is considerable, the outer fragment being drawn downwards by the weight of the arm, forwards and inwards by the pectoral muscles, and it is at the same time pulled behind the inner fragment, and somewhat rotated upon its axis by the action of the serratus magnus and pectoralis minor muscles, which pull the scapula forwards and inwards, whilst the rhomboidei draw up its lower angle. The sternal end of the clavicle practically maintains its natural position. Crepitus is easily felt when the shoulder is pulled outwards or when the acromion is depressed.

Forcible transverse compression of the upper part of the thorax occasionally breaks both

collar-bones at the same time. The patient is then rendered particularly helpless as the use of both arms is impaired. Dyspnœa is a frequent and important symptom. Simultaneous fracture of both clavicles is not necessary or even usually fatal, but it is often complicated by other injuries. The patient must be kept in bed and in the position best calculated to relieve his difficulty in breathing, and it should be remembered that there is a marked tendency for the fragments to override.

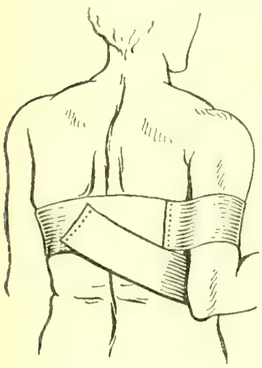


FIG. 1.—Sayre's method of treating a broken collar-bone; first step.

The *prognosis* is good in uncomplicated fractures, but the patient should be warned that there will always be some deformity at the seat of fracture, and that it may be a long time before he regains the power of swinging his arm round his head. In exceptional cases there will be shortening, and in a few cases I have seen non-union, though the arm has been useful. A broken collar-bone may be only a part of more severe injuries, and in such cases the surgeon should satisfy himself that there is no paralysis due to pressure upon the underlying nerves, that the axillary artery and vein are intact, and that the pleura and lung remain uninjured. The formation of exuberant callus is sometimes a very troublesome complication of fractured collar-bone, and it may be necessary to perform an extensive surgical operation to remove it. The fracture — especially in children—is often incomplete, and it is then very likely to be overlooked until the formation of callus makes the injury conspicuous, when the medical man is blamed because he has not applied a bandage. These incomplete or "greenstick" fractures are often seen in babies who have fallen out of bed, or who have been dropped by careless nurses, so that the history of the accident may be designedly misleading. For this reason I have long taught my pupils that when a baby or young child is brought with the complaint that it has pain and impaired movement at the point of the shoulder, it should always be treated as though the clavicle were fractured, even though no other signs of such an injury can be detected.

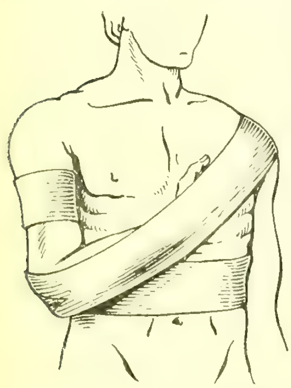


FIG. 2.—Sayre's method of treating a fractured collar-bone; second step.

Treatment.—Any method which keeps the shoulders well back and the injured arm at rest is sufficient treatment for a broken collar-bone. Sayre's method (Figs. 1 and 2) is the one usually adopted in this country. Two pieces of stout strapping are taken, measuring 3 inches in width by 5 feet in length. One piece of strapping is loosely stitched with the sticky side outwards, round the injured arm opposite the insertion of the deltoid. An assistant is directed to draw the shoulder downwards and backwards to stretch the clavicular fibres of the pectoralis major, and to reduce the displacement of the outer fragment of the clavicle. As soon as this is done the surgeon carries the strapping backwards round the body so that the sticky side adheres to the skin (Fig. 1). He then stitches the end, which passes between the side and the arm to the encircling band in the middle of the back. The elbow of the patient is then brought forwards so that the hand lies flat upon the opposite shoulder, and the second piece of strapping (Fig. 2) is carried obliquely across the sound shoulder in such a manner that the point of the elbow on the injured side is received into a slit made in the strapping, whilst the hand and arm are also covered. A flannelette bandage is applied over the strapping to keep everything in place. The position of the arm is an important factor in correcting the displacement, and the more the outer fragment is displaced downwards, the more necessary is it to keep the upper arm vertical (Fig. 3); if the displacement is inwards the upper arm must be placed well across the chest (Fig. 4), whilst if the forward displacement is the most marked, the upper arm must be placed as horizontal as possible (Fig. 5).

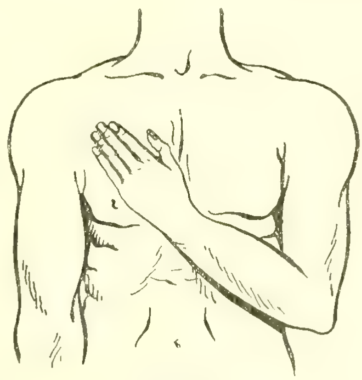


FIG. 3.—Diagram to illustrate the vertical position of the upper arm necessary to obtain the best results when there is a downward displacement of the outer end of a broken collar-bone. (Chiene and Dobie.)

Sayre's method is unsuited to men who have

much hair upon their chests, and it is badly borne by ladies with delicate skins. It is often convenient, therefore, to employ one of the

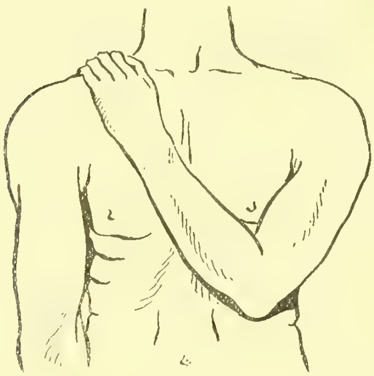


Fig. 4.—Diagram to illustrate that the upper arm must be placed well across the chest when there is much inward displacement of the outer end of a broken collar-bone. (Chiene and Dobie.)

two following methods :—(1) Lay the tail of a bandage on the hand (Fig. 6), carry it down the back of the forearm, round the elbow, up the upper arm to the tip of the acromion (it is too far forward in the diagram), across the back to the opposite axilla, across the forearm, round the elbow again, up the upper arm, then round below the deltoid and across the back to the opposite shoulder, repeating these turns as often as may be necessary. Last of all, fix the arm to the side by a few circular turns of the bandage. A layer of boracic lint or cotton wool dusted over with zinc oxide is to be placed between the arm and the trunk to prevent skin touching skin. (2) The "first-aid" method is often serviceable (Fig. 7). A square yard of linen is cut diagonally to make two triangular bandages, and a small pad of cotton wool dusted over with starch powder or oxide of zinc is placed in the axilla. One of the triangular bandages is then placed beneath the arm of the injured side in such a manner that the point of the triangle lies behind and on a level with the elbow, the arm being bent and the tips of the fingers resting upon the opposite arm. The upper end of the long side of the triangular bandage lies upon the sound shoulder, and the lower end is brought upwards over the injured arm and beneath the injured armpit until it can be tied to the upper end on the sound shoulder. The second bandage is then folded "shawl-wise" to make a narrow bandage, which is carried round the body to keep the injured arm at rest. For children it is often sufficient to put a sling round the wrist and to dress the patient with the injured arm inside the clothes. In the case of a lady, where it is important to obtain union with the least possible deformity, the patient must be content to remain in bed, flat upon her back, and with only a very narrow pillow under her head, the arm being fixed in a sling or by a plaster of Paris dressing. But even then the tilting of the scapula directs the outer end of the clavicle downwards, and somewhat rotates it upon its own axis, so that it is almost impossible to keep the ends of an oblique fracture of the shaft of the clavicle in perfect apposition. A fortnight in children and three weeks in adults is generally sufficient to ensure good bony union in simple cases of broken collar-bone, but this time is insufficient to allow of perfect consolidation, and a man who has to do manual labour should not be allowed to resume work for five weeks.

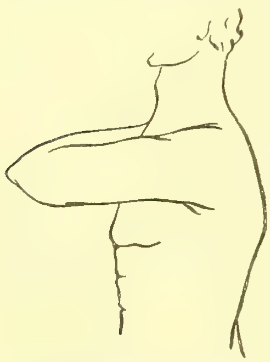


Fig. 5.—Diagram to show that the upper arm must be placed as horizontal as possible when there is much forward displacement of the outer end of a broken collar-bone. (Chiene and Dobie.)

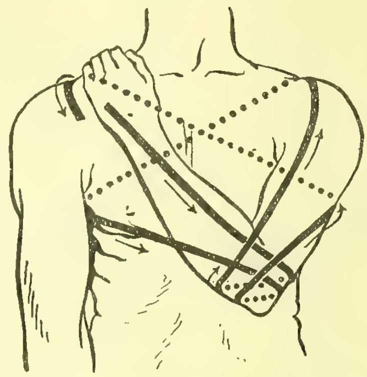


Fig. 6.—Bandage for a broken collar-bone when no strapping is used.

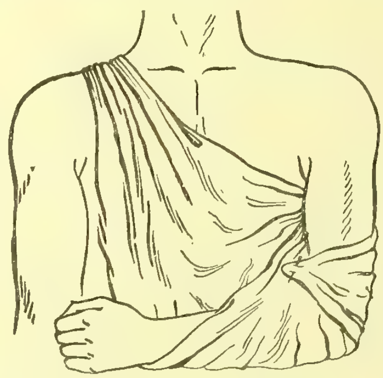


Fig. 7.—The "first aid" bandage for a broken collar-bone. (Manual for the R.A.M.C.)

Multiple fractures, double fractures, and complicated fractures of the clavicle, inasmuch as they are always produced by greater violence,

require the patient to be kept in bed for their satisfactory treatment.

Fracture of the sternal end of the clavicle is so rare an accident that only thirty-one cases are recorded—twelve due to muscular action, sixteen to violence, and three in which the cause was not stated. It is probable that in every case the accident has been rather a separation of the epiphysis than a true fracture of the sternal end of the bone. The treatment is the same as for a fracture of the shaft of the clavicle.

Fracture of the outer end of the clavicle is considered under the article "Shoulder Joint."

The Scapula.—Fractures of the scapula are not very common, but they take place at any part of the bone, because they are nearly always caused by direct violence. Fractures of the coracoid process and of the glenoid cavity are described in the article "Shoulder Joint." There remain to be considered fractures of the body of the scapula and of the acromion.

The *body of the scapula* may be broken by direct violence at the middle and at the upper or lower angle, or the fracture may be star-shaped. The exact nature of the injury may be very difficult to determine, as it is often associated with serious injuries elsewhere which prevent any thorough examination of the patient. There is often much swelling due to extravasation of blood; it is not always easy to obtain crepitus, and the effusion of blood in the connective tissue beneath the muscles causes a sensation which may be mistaken for crepitus; the scapula lies beneath a mass of muscle or adipose tissue in muscular and fat patients. When the body of the bone is broken, however, it is possible to discover a point where the local pain is greatest, and there is generally impaired movement of the arm when it is thrust forwards or horizontally. There may be considerable displacement when the upper or lower angle of the bone is broken, due to the action of the levator anguli scapulæ and of the serratus magnus muscles respectively.

The *prognosis* is good except in cases complicated by other injuries. The arm should be supported in a sling for four or five weeks, and adhesive plaster should be applied to the injured side of the back in the manner best calculated to keep the bony fragments at rest and in apposition. The patient should be kept in bed if there has been more than very slight injury to the soft tissues.

True *fracture of the acromion* is rare, although scapulæ with separated acromial processes are found in every pathological museum, for the majority of the specimens are examples of separation of the acromial epiphyses. In a case of true fracture of the acromion there is a slight flattening of the shoulder with a gap in the bone at the seat of injury, caused by the deltoid drawing the outer fragment downwards. Crepitus is not obtained until the arm is raised sufficiently to bring the two broken ends together. Local pain and tenderness are well marked, and there is impaired movement of the arm, especially in abduction. Repair takes place by fibrous union if there is much displacement, though it may be bony if the broken piece can be maintained in good apposition.

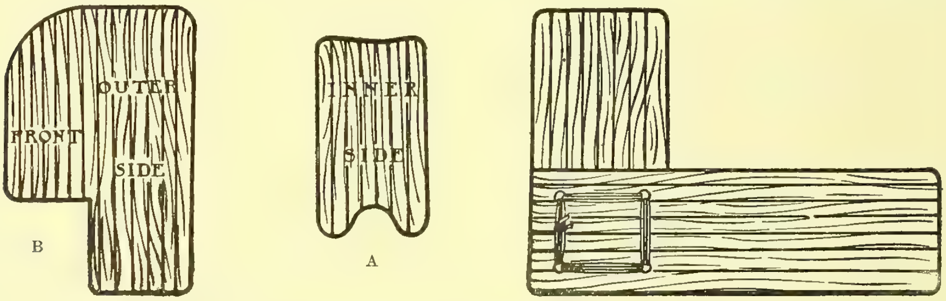
The *treatment* consists in placing a Stromeyer's cushion between the elbow and the side of the body, the arm being afterwards carried across the chest until the deltoid is completely relaxed. A plaster of Paris dressing passing round the chest and over the sound side shoulder fixes the injured limb. If it is undesirable to use plaster of Paris the arm should be supported by a large arm-sling to raise the elbow, and fixed by a bandage carried round the body, a gutta-percha or poroplastic cap being moulded to the shoulder. Passive movement may be commenced a fortnight after the accident, but the surgeon should not give too hopeful a prognosis, for many cases of apparent fracture of the acromion are associated with osteoarthritis.

The Humerus.—A broken arm is a very common accident at all times and in every station of life. Some of the varieties are considered under various headings (*see* "Elbow Joint," "Shoulder Joint"). It remains, therefore, to consider the fractures of the shaft of the bone. These fractures are either primary or secondary. The primary fractures are caused by direct violence, as by blows; by indirect violence from falls upon the arm and hand, or they may be the result of muscular action. Secondary fractures are not uncommon in persons affected with scirrhus of the breast when they are due to secondary deposits of cancer, which so weaken the bone that it is broken by a very slight degree of force. The primary fractures are generally simple, and they are either oblique, transverse, or comminuted, the amount of displacement varying with each variety being least in the transverse and most in the oblique form. The displacement varies, too, with the position of the fracture, so that surgeons have been inclined to classify fractures of the shaft of the humerus into fractures above and fractures below the insertion of the deltoid, owing to the traction exercised by this muscle. Although the nature of the injury is usually quite obvious, for there is pain, increased mobility, deformity, and shortening, it is sometimes difficult to recognise a broken arm, as in the case of a rickety child, where the fracture is often transverse without rupture of the thickened periosteum and the bone is already deformed by rickets.

The *prognosis* is good in the vast majority of cases, though the patient should be warned that there is some danger of non-union and of nerve complications. It may be assumed as a broad

rule for the prognosis of repair that the more marked the crepitus the more likely is there to be bony union, whilst great mobility at the seat of fracture, with only a little muffled crepitus, points to a complete separation of the two ends of the broken bone and to the intervention of a piece of the surrounding muscle or fibrous tissue. These conditions are likely to lead to the formation of a false joint unless the ends of the bones are exposed, the intervening tissue is removed, and the fragments are secured to each other by mechanical means. The musculo-spiral nerve is so closely attached to the humerus in the musculo-spiral groove, that it may be injured either at the time of the accident by the violence causing the fracture or by the spicules of broken bone, or, as is more often the case, by the nerve becoming involved in callus during the process of repair. As a rule the paralysis remains unnoticed until the splint is removed, when the patient complains of wrist-drop with impaired power of supination, accompanied by atrophy of the extensor muscles and loss of sensation in the skin supplied by the radial nerve. The application of electricity may be serviceable in some of the slighter cases, but unless improvement is marked and rapid it is advisable to cut down upon the humerus at the seat of fracture and free the nerve from its bony sheath.

The *indications for treatment* of a fracture near the middle of the shaft of the humerus are to fix the arm—whilst slight extension is kept up—in such a manner that the forearm may be flexed and slung without obstruction to the circulation at the elbow. This is best effected by applying a well-padded rectangular wooden splint along the inner side of the arm from the axilla to the finger-tips, with a Gooch's kettle-holder splint upon the outer side. Chiene recommends the following simple and satisfactory method:—"Take two Gooch splints, shaped as in Figs. 8 and 9, pad them well, and bind them on with a roller bandage. The notch at A (Fig. 9) is to avoid pressure upon the internal condyle, and the one at B (Fig. 8) fits the forearm, which is held at a right angle to the upper arm by means of a rectangular splint, also made of Gooch, as in Fig. 10, which reaches as far as the back of the wrist. The rectangular splint can be made easily by boring four holes and threading strong string, as in the Diagram." The bandage must be applied over the splint and to the forearm first with the elbow bent at a right angle. The actual seat of fracture should not be covered with the bandage. This bandage should end above the elbow. A second bandage should then be applied round the splint and humerus above the fracture, and whilst it is being applied the limb should be gently extended until it is of the same length as its fellow. The measurements are made from the lower and back part of the acromion, where a sharp edge of bone can be felt to the point of the olecranon. As soon as the splints have been applied, the hand and wrist are to be slung in a small arm-sling, that the weight of the forearm may help to maintain extension, and so diminish the tendency to shortening.



Figs. 8, 9, 10.—Simple splints for fractures of the shaft of the humerus made out of Gooch's "kettle-holder" splinting. (Chiene and Dobie.)

There is so real a danger of delayed union going on to the formation of a false joint after fracture of the shaft of the humerus, that it is best to keep the arm in splints until complete repair has taken place, that is to say, for three weeks in children, and a month or five weeks in adults. In cases of delayed union the arm must be again secured for a further period of five weeks, complete immobility being maintained by means of a poroplastic or plaster of Paris dressing, fixing the shoulder as well as the elbow. Some further operation is necessary when it is clear that the humerus is not going to unite in spite of prolonged rest. Many methods have been employed with success, but perhaps the most usual one is to expose the ends of the bone, to refresh them by removing the fibrous tissue which covers them, and by then uniting them in the best possible position either with stout silver wire or by means of steel screws. Bony union takes place in rather more than half the cases treated by operation.

Supracondylar Fracture. — Fractures of the lower end of the humerus are very numerous, but the majority involve the elbow joint and are discussed already (p. 45). A transverse

fracture of the humerus just above the condyles is not a very rare accident after a fall upon the outstretched hand, or, less often, from a blow upon the bent elbow. The line of fracture is oblique from behind forwards and downwards when the arm has been broken from indirect violence with the elbow straight; whilst the line of fracture runs obliquely from in front downwards and backwards when the accident has happened with the elbow bent. In either case there is well-marked shortening of the arm, the shortening disappearing when the arm is pulled upon, though it reappears as soon as the extension is relaxed. It is often impossible to bend the arm beyond a certain point, because the ends of the two fragments interlock.

The injury is soon followed by swelling, which makes a differential diagnosis difficult; but if the patient is seen directly, it is easy to distinguish a supracondylar fracture of the humerus from a dislocation backwards of the radius and ulna, by the fact that the internal and external condyles of the humerus and the tip of the olecranon maintain their normal relation to each other, that is to say, the point of the olecranon lies vertically below a line joining the condyles when the elbow is bent to a right angle, so that a ruler placed on the back of the humerus does not touch the tip of the olecranon when the arm is bent to this angle. In children this accident has to be distinguished from a separated epiphysis. "The crepitus," says Mr. Poland, "in separation of the epiphysis is more 'muffled'; the lower end of the upper fragment has a greater breadth than in fracture: the line of separation is nearer the end of the bone, and the anterior projection of the shaft of the humerus is on a level with the fold of the elbow, whilst in fracture it is usually above it: the anterior projection, too, in a separated epiphysis has a rounded extremity unlike the projection of the sharp end of a fracture." Many accidents attend a supracondylar fracture of the humerus in addition to impaired movement at the elbow. The pressure of the one end of the broken fragments may obstruct the circulation through the brachial artery, and may even cause gangrene, an examination of the radial pulse must therefore be made from time to time. The median nerve may be injured by the same pressure, or the ulnar and musculo-spiral nerves may, at a later time, be involved in the callus produced during the process of repair, whilst there is often permanent deformity of the elbow, which is most obvious in full extension of the arm known as cubitus varus.

Every variety of position, from extreme flexion to complete extension of the arm, has been advocated in the treatment of supracondylar fracture of the humerus. Prof. Chiene, than whom we could have no higher authority, recommends that the arm be placed vertically upon the chest wall, whilst the forearm is fully flexed. A figure-of-eight bandage is then applied to the elbow and the arm is bandaged to the side. The French surgeons maintain the arm in full extension, saying that by this means the fragments are kept in the best apposition. For my own part I prefer an intermediate course, and after reducing the fracture,

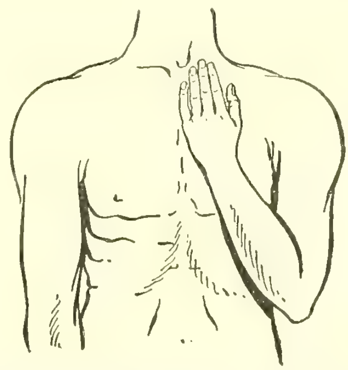


FIG. 11.—The position in which the arm is to be maintained in a case of fracture of the lower end of the humerus. (Chiene and Dobie.)

under an anæsthetic if necessary, I bend the elbow, put the arm midway between pronation and supination with the thumb uppermost, and place a well-padded rectangular splint of plaster of Paris along the inner side of the arm. The splint reaches from the axilla to the tips of the fingers and is kept in position by a roller bandage of linen, the arm being slung across the chest by a large arm-sling. Details of making and applying such a plaster of Paris case are given at vol. i. p. 362.

Passive movement should be commenced early, a point emphasised by Mr. Joseph Bell, who teaches that fractures near the elbow in young people should be kept quiet for a period corresponding to the age of the child, a day for each year, *e.g.*, a child of five years for five days, of ten years for ten days. Slight movements of flexion and extension are carried out at the elbow joint, care being taken that no movement is allowed at the fracture, and that no pain is caused. Voluntary movements may be allowed at the end of a month, but if there is much swelling or stiffness the arm should be shampooed daily from the twentieth day.

Intra-articular fractures of the condyles have already been considered (p. 45), but a portion of the inner condyle may be broken off without injury to the elbow joint. Such an epitrochlear fracture, or fracture of the internal epicondyle, may be the result of a direct injury to the inner side of the humerus, or, less commonly, to muscular action or a fall upon the hand. It occurs chiefly in young adults, and is often of the nature of a separated epiphysis. I have recently had two cases under my care, both occurring as the results of accidents in the foot-

ball field. The detached fragment is dragged downwards and either forwards or backwards by the flexor muscles which are attached to it, and by the pronator radii teres. There is much loss of function, passive movement is restricted, and the detached piece of bone can be moved independently. The accident may be associated with dislocation of the elbow: the ulnar nerve may be injured either by direct pressure of the condyle, or by the formation of callus; or "the carrying angle" of the arm at the elbow may be lost as the result of adduction of the fore-arm.

The treatment consists in semi-flexion of the elbow, wrist, and fingers to relax the flexor muscles; the epicondyle is then to be replaced as far as may be possible, being kept in position by a pad and strapping, and an anterior angular splint of plaster of Paris or metal is applied to the arm and forearm. The hand and wrist are alone slung.

Passive movement should be begun on the seventh to the tenth day, but it must be very gentle; the splints may be dispensed with upon the fourteenth day, when they are to be replaced by a sling. The detached piece of bone must be resected if the elbow be dislocated and the epicondyle prevents reduction; whilst the existence of nerve symptoms would lead the surgeon to expose the ulna at the seat of injury.

The Radius and Ulna.—Both bones of the forearm may be broken by direct violence when the fracture is at the same level in the two bones; by indirect violence, when the radius is usually broken at a higher level than the ulna; or by machinery accidents, when the bones are often broken at several different places after the arm has been drawn round a revolving wheel. Greenstick fractures of both bones are often seen in children who have fallen upon their hands.

Except in greenstick fractures there is usually no difficulty in recognising the nature of the injury, for the pain, deformity, loss of power and crepitus, are sufficiently characteristic. The prognosis is good, but the surgeon must bear in mind that in no other fracture is gangrene so common and so insidious, that impairment of supination and pronation frequently occurs, and that delayed union which may end in non-union may follow any imperfect fixation of the limb. The impairment of movement is due to several causes. It is sometimes caused by a rotatory displacement of the upper part of the radius, when the bone is broken above the insertion of the pronator radii teres, for the upper fragment is then supinated by the biceps, whilst the lower part of the bone is maintained in a semi-prone position. The power of full supination is lost if the bone heals in this position. In oblique fractures there is a good deal of overriding of the ends of the bone, and there is often some angular displacement. Crossed union of the broken ends of the bones may therefore easily occur, that is to say, the end of the radius may become adherent to the end of the ulna and *vice versa*, or the interosseous space at the seat of fracture may be filled with callus, and the usefulness of the limb is much lessened.

The *treatment* consists in carefully reducing the fractures by extension and counter-extension of the arm, care being taken that the interosseous space is maintained at its greatest width, *i.e.* in full supination when the fracture is in the upper third of the bones, and midway between supination and pronation when the bones are broken at any point in the lower two-thirds of the arm.

When the bones are broken in the upper third the arm is put up in full supination by means of a plaster case. The plaster case is made by taking a pattern in paper (see Fig. 12) from the sound side, in such a way that the splint will extend from the armpit, along the back of the arm, over the elbow bent at a right angle to the finger-tips. The pattern is laid upon a piece of ordinary house flannel of two thicknesses, over which a piece of lint is placed. The house flannel and lint are cut to the pattern, the inner lining of lint being sufficiently large to allow of its being wrapped over the edges of the flannel. The flannel is saturated with plaster of Paris paste, made by mixing one pound of freshly-burnt plaster of Paris with one pint of water, the excess of plaster being wrung out of the flannel. The lining of lint is then put in, and the case is bandaged to the arm, which must be held firmly until the plaster has set, that is to say, until the case gives a ringing metallic sound upon being struck. The arm is suspended by a sling reaching from the hand to the elbow. When the bones are broken in the lower two-thirds the same care must be taken to set them accurately, or if the fracture be greenstick, the bones must be straightened forcibly, but instead of the plaster case, two well-padded, straight wooden splints must be applied. The arm is held midway between pronation and supination, so that the thumb looks uppermost, for in this position the radius and ulna lie parallel to each

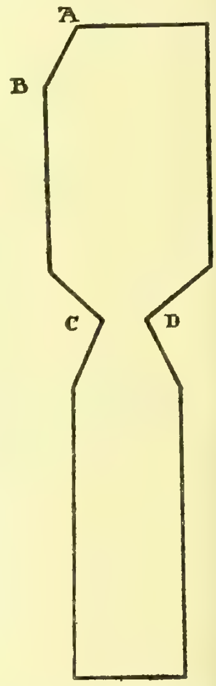


Fig. 12.—Diagram of a pattern for a plaster of Paris case for the back of the left arm. The piece AB is cut away for the axilla. The indentations at CD are to allow the elbow to be bent at a right angle.

other and at some distance apart, the splints are applied to the front and back of the forearm, and reach from the elbow to the back of the wrist, and from the bend of the elbow to the end of the metacarpal bones respectively. Each splint should be an inch wider than the transverse diameter of the arm to prevent the two bones being squeezed together by the bandage, but not wider, or movements will be allowed between the splints. The two splints are fixed in position by a strip of adhesive plaster applied spirally, a bandage is then put on, and the arm is supported by a large arm-sling, reaching from the elbow to the hand. Care must be taken that the inner splint does not press into the bend of the elbow, and the arm must be examined from time to time to see that gangrene is not taking place, for as the onset is sometimes painless, the statements of the patient are quite unreliable upon this point.

The arm should be kept untouched in splints for a fortnight. It may then be taken down and passive movements of the wrist and elbow may be made daily, the splints being afterwards reapplied until the end of the third or fourth week, when the bones should be strong enough to allow the arm to be supported in a sling only. Unless the patient has to use his arm for especially laborious work he may be allowed to resume his occupation at the end of the sixth week from the injury.

The Ulna.—Fractures of the olecranon and of the coronoid process of the ulna are considered elsewhere (p. 47).

Fracture of the shaft of the ulna is generally caused by a blow upon the arm, more rarely it is the result of indirect violence, and occasionally it has been caused by muscular action. In children the fracture may be subperiosteal and incomplete. The special dangers attending the injury are the probability of the fracture becoming compound owing to the subcutaneous position of the shaft of the ulna and the tendency of the radius to become dislocated forwards, outwards, or least often backwards when the ulna is broken in the upper third. The dislocation is usually an immediate result of the injury, but it may be secondary to non-union of the broken ulna, or to the formation of exuberant callus during the process of repair. The lower fragment is drawn towards the ulna by the pronator quadratus, and is often displaced backwards, forwards, or laterally by the injury causing the fracture. The upper fragment, too, may be displaced either inwards or outwards, for there is a little lateral movement at the elbow, but the main displacement is forwards. The injury is not difficult to recognise when it occurs in the subcutaneous part of the bone, whilst the limited flexion, pronation, and supination with pain felt most severely at one spot, enables a diagnosis to be made when the fracture has occurred in the upper third of the bone.

Care must be taken in setting the fracture to reduce the lateral displacement of the lower fragment of the ulna towards the radius, and not to overlook any dislocation of the head of the radius. The lateral displacement is remedied by pressing the fingers and thumb between the two bones. The arm is then bent to a right angle, and is secured in a plaster case moulded to the arm and forearm in the manner already described. The arm is then slung in a large arm-sling for three weeks.

The prognosis is good, though the excessive formation of callus sometimes limits the movements of the elbow after the fracture of the ulna in the upper third. When the fracture is incomplete the radius is a sufficient splint, and the arm need only be slung. The early and gentle application of massage from the fourth day onwards is often serviceable in reducing the pain and swelling in these cases of greenstick fracture. When the injury is associated with a dislocation of the radius the elbow must be maintained at an acute, instead of at a right, angle, passive movements being commenced at the end of a fortnight.

Fractures of the Radius.—Fractures of the head and neck of the radius are considered on p. 48, whilst Colles' fracture is discussed under "Wrist Joint."

Fractures of the shaft of the radius alone are less common than fractures of the ulna, but like them are usually caused by direct violence. The displacement varies according to the position of the fracture: when it is above the insertion of the pronator radii teres, and below the insertion of the biceps, the upper fragment is rotated outwards, whilst the lower fragment is rolled inwards; but when the fracture is in the lower third the upper fragment is drawn forwards by the biceps, and is rotated internally by the pronator radii teres, whilst the lower fragment is pulled inwards by the pronator quadratus and the supinator longus. The result, therefore, is often an angular deformity. The diagnosis of fracture of the shaft of the radius is easily established, if it is complete and unimpacted, by placing the fingers on the head of the radius, which can always be felt behind and directly below the external condyle of the humerus. If the radius be broken the head will not move when the hand and arm are gently pronated and supinated.

The treatment is the same as for fracture of both bones of the forearm (see p. 356), *i.e.* full supination when the fracture is above the insertion of the pronator radii teres, semi-pronation in all fractures of the middle and lower thirds. Repair takes place in a month, but the patient should be warned that pronation and supination may be impaired. Fractures of the various carpal bones are considered under the heading "Wrist Joint."

The Metacarpal Bones.—Fractures of the

metacarpal bones are said to form only about one per cent of all the fractures occurring in the human body, the third and fourth metacarpals being broken more often than the first. The

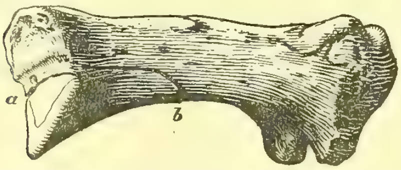


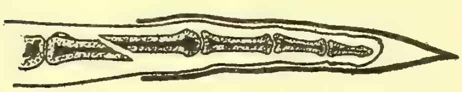
Fig. 13.—The metacarpal bone of the thumb to show the line of injury in Bennett's fracture of "the stave of the thumb."

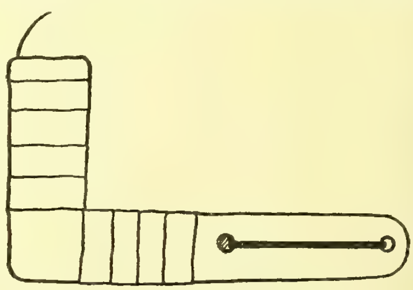
fractures are caused either by direct violence, as in fighting, the falling of a weight, or the kick of a horse, much more rarely by indirect violence, as by hyper-extension or over-flexion of the bones. Although complete fractures are the most frequent, it is by no means uncommon to see fissured fractures, and fractures where only a part of the bone has been torn off. The complete fractures are most common at the point where the bone is thinnest, that is to say, just above the middle. Prof. E. H. Bennett has recently drawn attention to a form of incomplete fracture which is not unusual in the metacarpal bone of the thumb, where after a fall upon the palm of the hand a fracture is caused which passes obliquely through the base of the bone (Fig. 13), detaching the greater part of the articular facet with that piece of bone supporting it which projects into the palm of the hand. The amount of displacement in this form of fracture is slight, but it consists in a subluxation of the thumb backwards. The displacement in the complete fractures is characteristic, unless it is masked by the swelling, for the proximal fragment is fixed whilst the distal end of the bone is tilted by the flexors of the fingers so that it projects upon the dorsum of the hand. Much more rarely the displacement of the distal fragment is into the palm of the hand, or even laterally. When a metacarpal bone is broken local pain, which is increased when pressure is made upon the head of the metacarpal, are sufficient to establish the diagnosis in cases of complete fracture. It is more difficult to recognise the injury when only a small piece of bone is broken off, but a radiograph will determine the nature of the injury.

The *treatment* consists in securing the hand for a fortnight to a well-padded Carr's splint, which can be made easily by screwing six inches of a broom handle obliquely on to the end of a straight wooden splint, so that the radial side is longer than the ulnar, the injured arm being afterwards suspended in a large arm-sling. A simpler plan is to bandage the closed fist over a pad of wool of sufficient size to completely fill the fist, but I do not think that this method is either so comfortable or gives such good results. In oblique fractures, where the surgeon considers it necessary to employ extension, a rectangular splint should be applied to the arm, so that it reaches well beyond the tips of the fingers; sticking plaster may then be applied to the injured finger, as in Fig. 14; the plaster is attached to a piece of elastic tubing, which is then carried through a hole in the splint and fixed to the back of the splint (Fig. 15).

There is often a little shortening after an oblique fracture of a metacarpal in spite of all treatment, but it is not of much importance, as it does not interfere with the movements of the hand. Repair is often very slow, though non-union is a rare result, and the formation of exuberant callus sometimes hinders the free movement of the fingers if it displaces the long flexor tendons. The prognosis of a simple fracture of the metacarpals is good, therefore, as regards union, but in many cases in Bennett's fracture of the metacarpal bone of the thumb there is often a lame and useless hand for many months after the injury.

The Phalanges.—The proximal and ungual phalanges of the fingers are more often broken than the intermediate ones, and as the injury is





Figs. 14, 15.—Method of applying a splint in a case of oblique fracture of a metacarpal bone. (Chiene and Dobie.)

close to the head, the distal end is carried forwards into the palm of the hand, and the accident may be mistaken for a metacarpo-phalangeal dislocation.

The displacement and the increased mobility at the seat of fracture, combined with the sharp always the result of direct violence, it is often compound. The line of fracture in the distal phalanges may be vertical. In simple fractures, and in every case where the thumb is injured, attempts should be made to ensure repair, but in compound comminuted fractures of the ungual

phalanges it is better to amputate at once, because union will probably result in a stiff finger-tip anchylosed at an awkward angle after very prolonged treatment. It is usually sufficient in simple fractures to adjust the fragments and keep the finger straight by applying a well-padded splint of wood, millboard, or gutta-percha, reaching along the palmar surface from the wrist to the finger-end. Passive movements may be commenced on the tenth day.

THE PELVIS.—Much ingenuity has been displayed in constructing elaborate classifications of the various fractures to which the pelvis is liable. But it is sufficient for a practical work to divide them into simple fractures and complex, the simple fractures being those in which a portion of the bones are separated without other serious injury; the complex fractures being

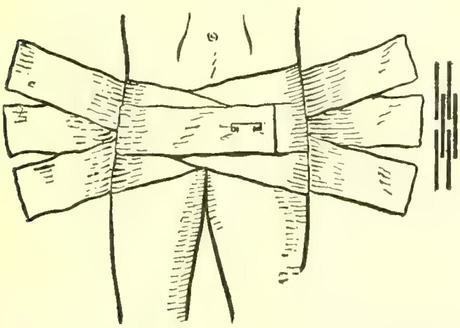


FIG. 16.—Method of applying a Scultetus' many-tailed bandage in a case of fractured pelvis. (Chiene and Dobie.)

associated with dislocations of the bones upon each other, or of the femur upon the ilium, with injuries to the contained viscera and urethra, or with epiphyseal displacements. A fractured pelvis is usually caused by some crushing force, as in railway accidents, when the patient has been caught between a moving train and the platform, or between the buffers of two carriages; or by a fall of earth, stone, or coal, as in mining or quarry accidents. The pelvis has been fractured occasionally by indirect violence, as in those cases where a patient has fallen upon his feet, and the force has been sufficient to drive the head of the femur through the acetabulum, thus splintering the innominate bone.

The *signs* vary with the nature and severity of the injury, but there is always pain and loss of power in the lower extremity. When the innominate bone is broken there is increased mobility at the seat of injury, and crepitus can be obtained by gently rocking the pelvis. Injuries to the ischium can be readily explored through the rectum or vagina whilst the tuberosity is moved beneath the skin. Rotation of the femur will cause crepitus when the acetabulum is broken.

The special dangers attending fractures of the pelvis are rupture of the urethra, laceration of the bladder either within or outside the peritoneum, tearing of the rectum, and injuries to the spinal cord, the larger nerve trunks of the lumbo-sacral plexus, the iliac arteries, or the iliac veins. Laceration of the membranous portion of the urethra is the most usual complication of fractures and separations at the pubes. It is of such importance to treat this injury at once, and before there has been any extravasation of urine, that a catheter should be passed in every case of fracture at the front of the pelvis, and if it is impossible to reach the bladder an external urethrotomy should be performed.

The *treatment* necessarily varies with the nature and severity of the injury, but it always requires that the patient should be kept in bed for a lengthened period. A many-tailed bandage, made in two layers and folded as in Fig. 16, is often a sufficient support. A loop of bandage should be passed under each thigh and pinned to the lower limit of the many-tailed bandage to prevent it slipping upwards. In bad cases double extension with the legs apart must be applied, and in all cases a tripartite mattress (Fig. 17), the central portion of which may be removed for the use of the bed-pan, is a most useful addition to the comfort of the patient, and it is indispensable when a fracture of the sacrum has caused paralysis of the lower extremities, of the bladder, and of the rectum. A patient may be allowed to get about on crutches in the simple cases at the end of six or eight weeks, his pelvis being protected by a poroplastic splint.

The prognosis varies greatly. The injury is often fatal, either at once from the shock and from the important structures involved, or after a long period of illness from the profuse suppuration of the connective tissues which are so abundant within the pelvis. Even in the least

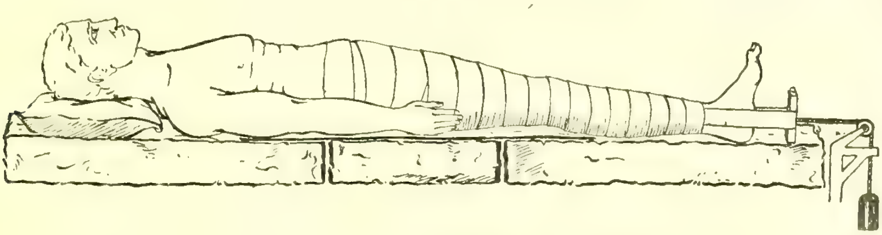


FIG. 17.—A tripartite mattress for use in fractured pelvis. (Chiene and Dobie.)

serious fractures, where bony union has taken place in six or eight weeks, there is often an impairment of movement, and the patient remains lame for life.

The Femur.—Fractures of the surgical neck of the femur, including the intra-capsular and extra-capsular fractures, impacted and unimpacted, are considered under the article "Hip-Joint," whilst in the same manner fractures of the lower end of the femur, involving or near the knee, will be discussed under the heading "Knee-Joint."

Fractures of the shaft of the femur are divided conveniently into those which occur below the trochanters and those of the middle third. They occur both in children and in adults, but in adults they are seen more often in men than in women.

Fractures in the upper third of the shaft of the femur are nearly always the result of indirect violence, though they are due occasionally to muscular action. They are always very oblique and are often spiral, but in children a fracture through the shaft of the femur may be transverse or of the greenstick variety.

The *signs* of a subtrochanteric fracture are sufficiently characteristic to leave very little doubt as to its nature in the majority of cases. The pain deformity, unusual mobility at the seat of fracture, and the crepitus are well marked, whilst there is much shortening. The displacement is considerable, and is a troublesome factor in treatment. The short upper fragment is tilted forwards at a varying angle to the pelvis by the action of the psoas and iliacus muscles, and is at the same time everted and drawn outwards by the external rotator and gluteus minimus muscles. The lower fragment is drawn upwards by the rectus and hamstrings, whilst it is rotated outward by the weight of the foot and the adductors. The direction of the obliquity often causes the lower fragment to lie along the inner side of the upper fragment.

The *indications for treatment* are to approximate the two ends of the bone, and to maintain the lower part of the shaft in an abducted position. These indications are followed by flexing the thigh, by traction, and by allowing it to lie somewhat upon its outer side during the process of repair. A Hodgen's splint (Fig. 18) is therefore the most satisfactory appliance for subtrochanteric fractures. It consists of two rods of iron slightly bent at the level of the knee, the two bars being connected at the lower end by a straight bar and at the upper end by a curved one. The splint reaches from the anterior superior spine of the ilium to three inches below the external malleolus on the outer side of the leg, and from the adductor longus tendon to an equal distance below the internal malleolus on the inner side of the limb. It is applied to the front of the leg and thigh, so that the limb is slung between the lateral rods by a series of strips of house-flannel which overlap each other, the ends of each strip being separated, stitched, or pinned round the sides of the splint. Extension is made by a weight,

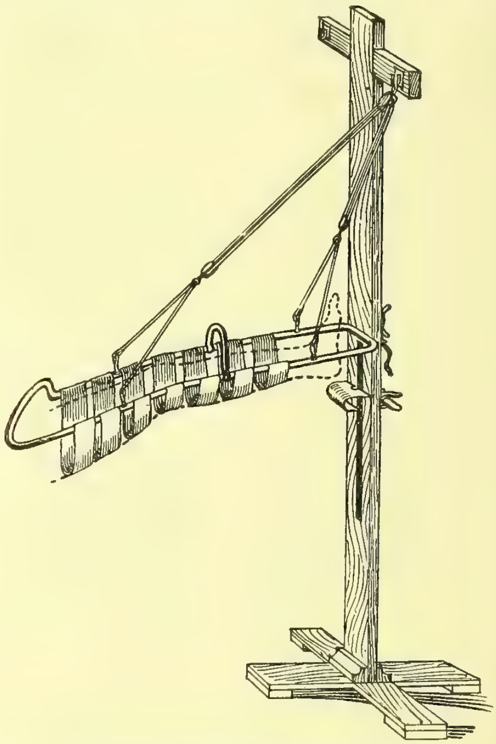


Fig. 18.—A fractured thigh treated by Hodgen's splint.

pulley, and strapping, in the manner to be described immediately, and the whole apparatus is slung at a convenient angle, as is seen in Fig. 18. Under many conditions it is inconvenient or impossible to use a Hodgen's splint, and recourse must then be had to the double inclined plane (Fig. 19). The angle should be as obtuse as possible, and only so much bandage should be applied as is sufficient to keep the limb at rest in a good position. The splint should be kept on for six weeks or two months, the thigh being massaged after the twentieth day. When the splint is removed the patient must be careful of his leg for some time, as it is not unusual for the thigh to be refractured as the result of a false step or a slip. A plaster of Paris case may be applied to the thigh for a month after the splint has been removed, the patient learning to walk in the meantime upon crutches. In every case he must be warned that the shortening may be so considerable as to cause him to limp, but there is usually good bony union. In cases of angular union, and when the limb is greatly crippled, the question of resection of the end of the bone may be reasonably entertained. The surgeon must, of course, deal with each of these cases on its merits, but unless he be skilful, experienced,

and in a position to carry out the operation without any chance of suppuration, he had better not undertake it even in the most favourable case.

Fracture of the Middle of the Shaft of the Femur.—Fracture of the middle of the shaft of the femur is a common accident both in adults and in children, either as a result of indirect violence or less frequently from blows upon the thigh or the passage of a wheel over it. The line of fracture is usually oblique from before backwards, so that the lower fragment passes behind the upper, which may be so sharp as to perforate the muscle and skin, thus rendering the injury compound. The obliquity may be so great as to make the line of fracture almost vertical, or it may become spiral by following the arrangement of the fibres of the osseous tissue at this part of the femur. The union of various lines of fracture may detach large splinters of bone, though the fracture is not truly comminuted. On the other hand, greenstick, subperiosteal, and transverse fractures are by no means rare in children, and true comminuted fractures may occur in them, as a result of scurvy, without any appreciable violence.

The *signs* of a fractured thigh are generally sufficiently distinct. There are pain, loss of power over the limb, abnormal movement at the painful spot, and crepitus, whilst the injured leg is materially shorter than the sound one, and is very completely everted. In making comparative measurements of the length of the two limbs care must be taken that the patient is lying with his two iliac bones upon the same level, for inaccurate results will be obtained if the pelvis be tilted. The measurements are made from the anterior superior spine of the ilium to the tip of the external malleolus. The diagnosis is often extremely difficult in children with incomplete fracture, and the nature of the injury is often entirely overlooked. The prognosis is good in simple fractures of the middle of the shaft of the femur, but the surgeon should remember that delayed and non-union are not uncommon, that it is the most difficult thing in the world to get union without shortening when the fracture is very oblique or comminuted, and that a passive synovitis of the knee takes place in nearly every fractured thigh, even though the bone is broken in the upper or middle third.

Treatment.—There are very many different methods of treating a simple fracture of the thigh, but here, as always, the simplest is the best, and the application of a long external splint with a weight extension will usually answer every purpose. In the first place, care must be taken in moving a person who has fractured his femur to prevent the simple fracture becoming compound, and this accident is the more likely to happen the nearer the injury is to the knee. The limb should therefore be immobilised by securing a long external splint and a shorter internal one to the injured limb. A lath or broom handle reaching from the armpit to the sole, with an umbrella or walking-stick along the inner side of the thigh, will answer this purpose temporarily, if they be secured with handkerchiefs tied round the groin, above the knee, below the knee, and at the ankle, the patient's legs being afterwards bound together for additional security. The patient will have to be kept in bed and nursed for several weeks, and he should therefore be placed at once upon a low and narrow iron bedstead provided with a good hair mattress, which is not likely to become uneven, and sand-bags should be placed upon either side of the leg until the surgeon is ready to reduce the fracture.

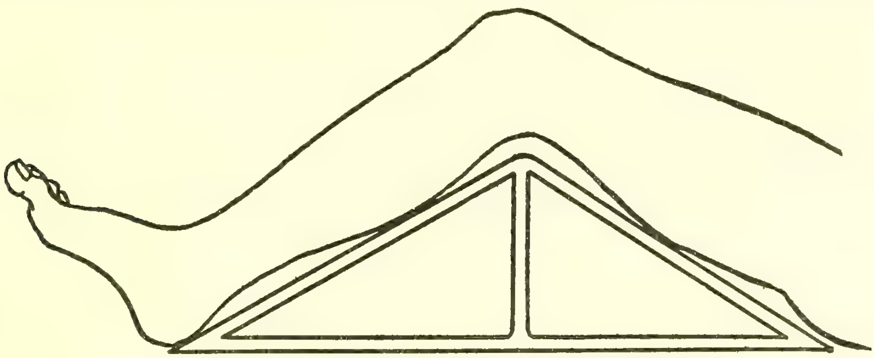


Fig. 19.—A double-inclined plane for use in certain fractures of the femur.

The leg and thigh should now be carefully washed, dried, and dusted over with oxide of zinc. A piece of stout moleskin strapping, $2\frac{1}{2}$ inches wide and about 5 feet in length, is doubled upon itself, and a square piece of wood, whose diameter is equal to the width of the ankle at the malleoli, is secured in the centre of the strapping by four drawing-pins. The strapping is warmed and applied to either side of the leg, care being taken that it reaches two or three inches above the knee, and that it does not stick to the malleoli, for which purpose the lower six inches of strapping upon either side of the leg

are rendered non-adhesive by guarding each strip with a second piece of strapping of the same width, so that the two adhesive surfaces are together. The two pieces of strapping running up the leg are kept in place by winding a second piece of plaster spirally round the leg. A cord is then passed through the centre of the wooden stirrup, which should lie quite square about three inches from the sole of the foot. A long external splint is now chosen, of such a length that one end lies comfortably in the axilla without exerting any pressure upon the armpit when the arm is by the side, whilst the external malleolus is opposite the centre of the hole in the splint which is made to receive it. The splint should have no foot-piece, but a flat cross-piece is screwed horizontally to the lower end to keep the splint steady. The whole splint must be well padded with tow, and it is then laid along the injured side of the patient, who is placed flat on the bed without any pillow under his head. Two assistants make steady extension upon the limb until the deformity at the seat of fracture disappears, the one taking hold of the thigh just above the knee and rotating it inwards, whilst the second assistant grasps the foot and ankle, which must also be rotated inwards to the same extent as the thigh. The surgeon secures the splint by applying a bandage round the foot and ankle in such a manner as to keep the foot at right angles to the leg, the bandage being afterwards carried up the leg as high as the tubercle of the tibia. A second bandage is then applied to the thigh above the seat of fracture, and a broad flannel bandage is carried round the chest and over the splint. The knee and the thigh in the immediate neighbourhood of the fracture are not bandaged, and it is best to apply the bandage to the foot and leg from without inwards to counteract the tendency to eversion. When a single outside splint fails to support the fracture it may be reinforced by a straight and well-padded wooden splint put at the back of the limb and reaching from the gluteal fold to the top of the calf. In other cases a piece of Gooch's "kettle-holder" splint may be cut to encircle the front and inner side of the thigh from the groin to the top of the patella. A small pulley is then fixed to the end of the bed, either by screwing it into a piece of wood fastened to the bed-foot, or by means of a standard with a movable arm standing just beyond it. The cord attached to the stirrup runs over the pulley and, for an adult, carries a weight of ten pounds. The foot of the bed is then raised about four inches by means of two blocks of wood, that the patient's body may act as a counterpoise to the traction of the weight. The injured limb is wrapped in a small blanket, a cradle is put over it, and the rest of the bed is made as usual. Attention must be paid during the after-treatment to the comfort of the patient, especially to any indication of chafing at the heel, the malleoli, or over the sacrum, and the surgeon should bear in mind that the effects of chronic alcoholism show themselves more readily after a fracture of the thigh than after any other fracture. The first effect of the weight is to overcome the muscular spasm, but as this lasts from twenty-four to forty-eight hours, sufficient time should be allowed to elapse before the lighter weight is replaced by a heavier one. In muscular subjects a much heavier weight will be required to secure a proper extension, whilst in children the extending force is generally calculated as two pounds for a child of two years old, increasing a pound for each additional year. The weight and pulley need constant attention, as the cord often jams and renders the extension useless.

In children, where the fracture is much less oblique than in adults, a plaster of Paris case applied to the front and back of the limb and an extension apparatus answer admirably, the Liston's splint being applied to the uninjured side for the purpose of making the child lie flat in the bed. Children who have incontinence of urine, very young children, and those who have both thighs broken, can often be treated satisfactorily without any splint, by keeping them flat upon their backs, and rigging up an extension apparatus consisting of a weight and pulley so situated above the bed that the thighs are flexed at a right angle with the body. Restless children who cannot be induced to lie straight and flat by any other means are best secured in a Bryant's or double Thomas's splint.

The duration of treatment varies with the age, weight, and general condition of the patient. Three weeks in bed is sufficient for a baby who cannot walk; for a child, a month with a further fortnight in a plaster of Paris case; but as age increases and the patient is heavier, a longer time is required, so that an adult will be four or five months before he can walk with ease. The wasting and the stiffness of the knee are the most troublesome factors in the convalescence, and these are combated satisfactorily by the employment of massage from the twentieth day onwards, or at a later period by the use of a hot-air apparatus. The splint may be permanently removed at the end of five or six weeks, but the patient should then be kept in bed until at the beginning of the seventh week he is allowed to get up with a plaster case upon his thigh. He must then learn to walk upon crutches, for too early walking upon a fractured femur causes bending of the bone, or it may again be broken.

The symptoms and treatment of inter-condyloid fractures of the femur, and of fracture of the inner and outer condyles are considered under the heading "Knee, Diseases and Injuries of."

Fractures of the Leg.—Fractures of the upper end of the tibia necessarily involve the knee-joint, and are described under the article "Knee-Joint."

Fractures of both Bones of the Leg.—

Fractures of the shaft of the tibia and fibula are the result of direct violence, when the fractures are either transverse or comminuted, and the two bones are broken at the same level; or they are caused by indirect violence, when the tibia is broken first, the fibula yielding afterwards and at a higher point. A forcible muscular contraction is occasionally sufficient to fracture the tibia, the fibula breaking subsequently when an attempt is made by the patient to bear his weight upon the injured limb. The tibia is especially liable to be broken by torsion, and in these cases the line of fracture is often spiral, or the lower fragment is comminuted whilst the upper fragment terminates in a sharp V-shaped end which often penetrates the skin, making the fracture compound.

The signs are usually so well marked that there is no difficulty in recognising the injury, though it is sometimes difficult to discover whether the fibula is broken as well as the tibia. The prognosis varies with the severity of the injury, except in children. Good bony union nearly always takes place in a simple transverse fracture where there is little or no displacement. But when the fracture is comminuted or oblique the repair is much less satisfactory, for it takes considerably longer, there is often impaired movement at the knee or ankle, and the patient may complain of rheumatic pains for many years afterwards. The course taken by a compound fracture which has become septic is still more tedious, and in unhealthy people is often dangerous. In children of two or three years old the simplest fracture requires as much care in its treatment as the most severe, for I have several times seen so intractable a form of non-union as a result of of simple fracture that amputation has been required.

Care during the first few minutes after a patient has broken his leg will often save months of trouble, for thoughtless bystanders often try to set a man upon his legs when he has fallen, or lift him so roughly or carelessly as to force the upper end of the broken tibia through the skin, thus converting a simple into a compound fracture. A doubtful injury to the leg should always be treated as a fracture, and the patient should be moved out of harm's way as gently as possible, some one supporting the injured limb and doing nothing else. An improvised splint should always be applied to the injured limb to steady it whilst the patient is being removed to his home or a hospital. The two parts of the broken leg are to be kept in the same straight line, and the injured limb is on no account to be allowed to hang down, nor should any attempt be made to drag off the boot or stocking. It is best to lay the leg on a pillow or on a folded coat, a handkerchief being tied round the leg and its support above and below the seat of fracture, or two lateral splints may be improvised out of a couple of walking-sticks, umbrellas, or even newspapers folded lengthwise.

The subsequent treatment varies with the severity of the injury and, perhaps more than any other fracture, with the school in which the surgeon was educated. Many surgeons content themselves in the simplest form of fracture with applying a plaster of Paris case at once; others keep the patient in bed a week to allow the swelling to subside and then employ a plaster case; whilst yet others, and I confess that my sympathies are with them, keep the patient in bed for a longer time and sling the leg in a back splint.

The plaster case consists of layers of house flannel shaped to fit the foot and leg (Fig. 20), and soaked in plaster of Paris in the manner described at p. 362, vol. i. An antero-posterior splint gives better support than the lateral ones usually recommended. The anterior splint is a narrow one reaching from the top of the patella to the end of the metatarsal bones; the posterior one is broad enough to support the leg both on its inner and outer sides, and it extends from the top of the ham to the web of the toes. Care must be taken whilst the splint is hardening to maintain the full length of the leg, to keep the foot exactly at right angles with the leg, especially to avoid eversion of the foot, and to see that the lower fragment is neither twisted nor tilted upon the upper fragment. The two parts of the plaster of Paris case are bound on to the leg with a figure-of-eight bandage applied before the plaster has set. If the plaster case is to be used at once it must be put on before the swelling has commenced, and the surgeon in charge of the case should himself hold the leg in proper position until the plaster has become thoroughly hard. He should remember that bullæ often form under the skin when the leg is broken, and that a case which fits accurately at first becomes sufficiently loose in the course of a week to allow of the fragments becoming displaced. He must therefore keep the patient under daily observation, and be prepared to apply a fresh case as soon as may be necessary. Massage may be commenced from the seventh day in simple cases, and should be particularly directed to the knee and ankle. The patient should be kept at rest for three weeks when both bones are broken, and he may then be allowed to walk upon crutches, the injured leg being kept in the plaster case for at least six weeks.

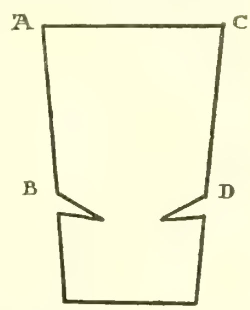


Fig. 20.—Diagram of a pattern for a plaster of Paris splint for the leg. The side AB corresponds to the length of the limb, AC to its circumference at the thigh and ankle respectively, the piece below BD being for the foot, and the indentations being for the ankle.

A certain amount of risk attends the immediate treatment of even a simple fracture by this method, and many surgeons therefore prefer to keep the patient in bed until the swelling has subsided before applying a plaster splint. The foot and leg must be kept steady and in a proper position for a week or more, and this is best effected by a back splint and suspension of the leg. For this purpose a well-padded metal back splint with a rectangular foot-piece is selected (Fig. 21). It should be sufficiently long to reach as high as the middle of the thigh, and broad enough to prevent any compression of the leg by the side splints and bandage. The splint must be bent to an obtuse angle opposite the knee to keep the joint in slight flexion, and thus to relax the muscles of the calf. The splint should be provided with two cross-pieces with perforations of sufficient width to allow an ordinary leather rug-strap to pass through the hole on each side, one cross-piece being situated just above the foot-piece, the other immediately below the knee. Two well-padded, straight wooden splints are also obtained, long enough to reach from the foot-piece to the knee, and about four inches in width. Provided with this apparatus, some small pads, a roll of strapping, bandages, safety-pins, a pair of rug straps, a "cradle," webbing straps with buckles, and a teacupful of starch paste, the surgeon proceeds to set the broken leg. The whole limb is shaved if necessary, and is thoroughly but gently washed with warm soap and water; it is dried and is dusted over with oxide of zinc. It is then raised from the bed by the surgeon, who takes a firm hold of the foot and of the calf, whilst an assistant slips the splint beneath it. The leg is then lowered on to the splint, the assistant putting a small pad beneath the tendo Achillis to prevent any pressure upon the heel. The foot is secured to the foot-piece by a roller bandage, one end of which is pinned into the padding on the inner side of the splint. The bandage is then carried between the padded foot-piece and the sole of the foot to the outer side, thence across the foot, round the back of the foot-piece, and again round the foot with a reverse. The surgeon now takes the foot-piece to which the foot is attached in his hands, whilst the assistant steadies the leg. Gentle traction is then made upon the foot until the displacement is reduced at the seat of fracture, and until the two fragments of the tibia are brought into good apposition. The foot and the lower fragment are also moved until the inner edge of the patella, the inner ankle, and the inner side of the great toe are in the same vertical plane when the patella looks directly upwards. A strip of adhesive plaster two inches wide is then carried spirally round the leg and splint just above the malleoli, a similar piece just below the knee, and a third piece round the thigh. A few turns of bandage with reverses are then put on at each of these places, leaving the fracture and the knee unbandaged, and a little starch paste is brushed over the bandages to keep them in position. The two lateral splints are applied to the leg and are kept in place by webbing straps, the cradle is put over the limb, and the splint is slung to it by the straps, so that it swings just clear of the bed.

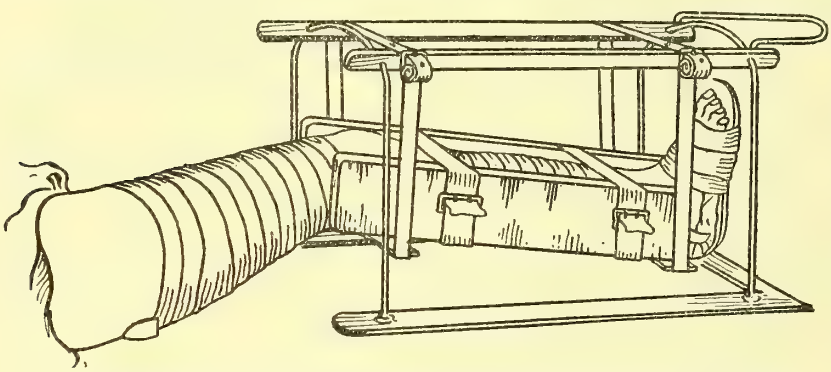


Fig. 21.—Fractured leg treated by a back splint with side splints, the limb being swung in a cradle.

Professor Chiene gives the following directions for making an improvised box splint when the more elaborate form here described is not available:—"Take two flat pieces of wood about half an inch thick, and between four and five inches broad, and long enough to reach from a little above the knee-joint to beyond the heel. A large sheet folded so that its breadth may be equal to the length of the splint is then prepared, and the splints are rolled up in it towards one another, beginning with one at either end of the sheet. Go on folding until the distance between the two splints, when vertical, may, along with a little padding, be just sufficient to enclose the limb. Pad well, and fix in position with three slip knots. Keep the foot at right angles to the long axis of the limb by means of elastic or a bandage, and place the leg upon a pillow. In oblique fractures of the tibia extension may be required to prevent shortening. In compound fractures it may be necessary to mould a splint to the leg, and rabbit wire netting folded into layers, to give it sufficient stability, with an old blanket as padding, can be fashioned into an excellent splint accurately fitting the injured limb."

When a box splint is used the surgeon must carefully watch the limb to see that the heel as

well as the toes touch the rectangular foot-piece, as even a little equinus is very troublesome to the patient when he begins to walk again. He must also make sure that the heel pad is effective in keeping pressure off the heel, which easily gets sore. A well-applied splint, too, is comfortable, and will prevent the spasmodic starting which is often a very painful accompaniment of fracture. The surgeon should have no hesitation in taking down the whole splint and re-adjusting the fracture, if there is the least real ground for complaint, within the first few days of the injury, and he must always be ready to readjust the bandages to accommodate any unusual amount of swelling. The splint may be abandoned on the twenty-first day in favour of the plaster of Paris case before described, but massage of the leg and ankle may be commenced a week earlier. The process of shampooing is particularly valuable in these cases, because the skin is apt to get dry and scurfy. It is better not to interfere with the bullæ, which are often large and numerous.

There are many cases in which it is impossible to get the fragments of bone into good apposition, and it is then justifiable to undertake a formal operation, exposing the ends, wiring them, and uniting the wound so that it heals by first intention.

Treatment of Compound Fractures of the Leg.—Compound fractures of the more simple variety should always be treated in the hope that they may heal aseptically, or at any rate with a minimum of constitutional disturbance. The surgeon shall first wash and disinfect his hands, and when a sharp fragment of bone protrudes through the skin it must be thoroughly cleansed with hot soap and water. It is then bathed in a solution of biniodide of mercury, which is first of a strength of 1 in 500, and afterwards of 1 in 1000, the biniodide being somewhat more satisfactory than the perchloride of mercury. The fragment of bone is afterwards replaced—and the skin covering the shin is so thin and elastic that a comparatively large piece of bone leaves but a small hole when it has been returned to its normal position. The skin, both in the neighbourhood of the wound and for some distance round it, must be cleaned and thoroughly disinfected by scrubbing it with a nail-brush, after lathering with soap and water. It is then washed with turpentine or methylated spirit, soaked for two minutes in a solution of biniodide of mercury in methylated spirits (1 in 500), and afterwards dressed with a piece of cyanide gauze wetted with a solution of 1 in 2000 biniodide of mercury. A layer of cotton wool is placed over the gauze, and the whole is kept in place by a bandage. The limb is then treated in the same way as a simple fracture, and, in a healthy person, it often heals without further trouble. It is better to enlarge the wound if there is any difficulty in reducing the fragment rather than bruise the skin and soft tissues by using violence. The fragment may be sawn off in extreme cases, and if there is much bleeding and laceration the patient should be placed under an anæsthetic and the parts thoroughly disinfected, the detached fragments of bone being removed, and the limb dressed in the manner just described. Severe compound comminuted fractures with extensive destruction of the soft parts and injury to the vessels and nerves require amputation.

Fractured Fibula.—The upper end of the fibula may be fractured by direct violence, as by the passage of a wheel over the leg, the tibia escaping injury; by indirect violence when the leg is so powerfully adducted as to put an extra strain upon the external lateral ligament, or by a sudden contraction of the biceps femoris muscle.

The signs of fracture are the altered position of the head of the fibula owing to the upward displacement caused by the biceps, and the impairment of function due to pressure upon, or injury to, the peroneal nerve at the point where it winds round the head of the bone.

The prognosis depends in great part upon the nerve lesions, which are either immediate or remote. Severe pain in the course of the nerve, with a burning pain referred to the foot, after an injury to the top of the fibula, is evidence that the nerve has been injured, whilst herpetic eruptions, ulceration, glossy skin, and the appearance of talipes equino-varus at a much later time, due to paralysis of the peronei and extensor muscles, would indicate that the nerve was seriously implicated, and would justify an incision in its course to ascertain the cause. Inasmuch as the synovial membrane of the tibio-fibular articulation sometimes communicates with the knee-joint, fracture of the upper end of the fibula is occasionally associated with some swelling and inflammation of the larger synovial membrane.

The treatment consists in relaxing the biceps to keep the two fragments in good apposition, and the leg should therefore be kept with the knee bent to a right angle for a fortnight or three weeks, though if an incision has to be made to explore the nerve there is no reason why the two fragments should not at the same time be united with silver wire. At the end of three weeks the patient may be allowed to walk with his leg and knee enclosed in a plaster case.

Fractures of the middle of the shaft of the fibula are generally caused by direct violence. They are either oblique, transverse, comminuted, multiple, or much more rarely incomplete. Simple fractures of the middle of the shaft of the fibula are sometimes very difficult to recognise, for the bone is well surrounded by muscle and the displacement is not great. Pain can be elicited locally by pressure, as well as by "springing" the fibula, that is to say, by hold-

ing the lower part of the leg steady whilst pressure is made upon the upper part of the outer side of the fibula. A skiagraph, however, will always make clear the nature of the injury. Here, if anywhere, the ambulatory treatment with massage may be employed. It consists in the application of a plaster of Paris case in two lateral portions. The foot must be kept at right angles, and care must be taken to see that the case continues to fit so long as it is used. Repair takes place in three weeks, but the patient may complain of neuralgic pain in the seat of injury. Fractures of the fibula within four inches of the external malleolus are considered under the article "Ankle-Joint," vol. i. p. 197.

Fractured Tibia. — Direct violence may fracture the tibia either at its upper end, when the line of fracture is transverse or more rarely vertical, or at any part of the shaft, when the fracture is usually oblique. The bone is sufficiently subcutaneous to enable a fracture to be detected easily if the patient be seen before the swelling has occurred, but at a later time the diagnosis is more difficult, since the fibula acts as a splint and there is very little displacement. The prognosis is good. The treatment consists in keeping the patient in bed with his leg on a rectangular splint until the swelling has subsided. He may then be allowed to go about on crutches after a plaster of Paris case has been applied.

Fractures of the Tarsus (fractures of the astragalus are considered under the heading "Ankle, Diseases and Injuries of"). — The os calcis is subject to several fractures. The bone may be crushed as a result of falls upon the feet, or severe injuries may be inflicted upon it by the forcible contraction of the powerful muscles attached to it; less often splinters of bone may be detached, or some of its bony prominences may be separated.

The foot is flexed at the ankle when the body of the bone is broken and the power of extension is lost; there is great pain in the tarsus, and crepitus can usually be felt. The width of the heel is increased, and the malleoli are nearer to the sole than on the sound side. The patient should be kneeling whilst the surgeon makes his preliminary examination. The diagnosis is often difficult, and the injury has been mistaken for a Pott's fracture and for a severe sprain. The prognosis is not very favourable, as some amount of flat-foot is nearly always left, and in severe fractures the ankle-joint may be involved. Treatment consists in keeping the foot and leg at rest in a plaster of Paris case for fully three weeks, massage being commenced at the end of a fortnight. When a violent contraction of the tendo Achillis has torn away a fragment from the posterior aspect of the os calcis it may be necessary to pin the fragment to the body of the bone, if it cannot be kept in place by flexing the knee and extending the foot.

The sustentaculum tali is occasionally torn off by forcible inversion of the sole of the foot. The accident is attended by pain, inability to walk, a sudden change from inversion to eversion of the foot, and shortening of the heel due to a slight displacement forward of the os calcis. Crepitus may be felt on the inner border of the foot. The prognosis is complicated by the subsequent tendency to flat-foot. The treatment consists in the application of a plaster of Paris case, with sufficient inversion of the foot to bring the two fragments together.

Fractures of the Metatarsal Bones.—The metatarsal bones are nearly always broken by direct violence, and it is said that the first and fifth metatarsals are more often fractured than the second, third, or fourth. The injury is either simple or compound, and the broken ends of the bone project beneath the skin on the dorsum of the foot. There is often so much swelling and bruising of the foot as to render the diagnosis difficult, though the localised pain renders the nature of the injury obvious.

In simple fractures the prognosis is good if care be taken to get union with the least possible displacement, but in compound fractures the severity of the injury may render amputation indispensable. The dirty habits of people liable to injury of the metatarsal bones make it necessary to be especially careful in disinfecting the feet when the skin is abraded, for the bruised tissues easily suppurate. A Cline's splint with the foot-piece at right angles applied to the outer side of the foot may be employed in these cases until the swelling has subsided.

Fractures of the Metatarsal Phalanges.—The phalanges of the toes are broken by direct violence, and the fracture, except in the great toe, is nearly always compound. The pain and crepitus render the diagnosis easy. The prognosis is good in simple fractures, but extensive suppuration and necrosis may follow a compound fracture unless it be thoroughly disinfected immediately after the injury.

A gutta-percha splint may be moulded to the inner side of the foot in fractures of the great toe, whilst in fractures of the phalanges of the other toes the dressings applied to the wound are generally sufficient to keep the fragments in good apposition until union takes place. Every effort must be made to preserve the great toe after fracture, but in the other toes, when there has been comminution with much bruising of the tissues, it is sometimes more profitable to amputate at once.

Fragilitas Crinium. *See* Trichorrhexis Nodosa.

Fragilitas Ossium. *See also* Achondroplasia; Pregnancy, Intra-Uterine Dis-

EASES (*Osteogenesis Imperfecta*); RICKETS (*Fœtal Rickets*).

Fragilitas ossium or *Osteopsathyrosis* may be defined as an unnatural brittleness of the bones. The terms are employed to denote two different conditions—(i.) An abnormal brittleness of the bones occurring as a symptom in consequence, for example, of tumours in the bones, disuse, senility, joint disease, cachexia of malignant disease, syphilis, rickets, insanity, locomotor ataxia, scorbutus—*e.g.* several fractures recorded by Guthrie in a child æt. 2½ years, who had scurvy; and lastly, phosphorus poisoning, as in two workers in a lucifer-match factory, each of whom had "at different times both thigh bones broken in a ridiculously simple fashion" (Dearden). In such cases there is rarefaction of the bones, which are light and porous, and which may show deformity from repeated fractures. (ii.) Idiopathic Fragilitas ossium, a separate and distinct condition, not secondary to any pre-existing disease. The brittleness here is not accompanied, so far as is known, by any constant local alterations, nor does the chemical constitution of the bones satisfactorily account for their fragility. The bones, though very brittle, do not tend to bend, but in consequence of numerous fractures they become much deformed. The bones, and especially those of the lower limbs and the ribs, are liable to fracture after very slight violence, *e.g.* while throwing a cricket-ball (Willet), from a slight fall or blow, or even from mere movement, say, of the limbs, as in Spurrell's case. The disease is often, though not always, hereditary, and several members of the family may be affected, as in Hunter's cases. The first fracture often occurs during childhood, and subsequently fractures take place repeatedly. Stanley quotes the case of a girl aged fourteen, who had had 31 fractures. Dent records 27 fractures in a man aged twenty-nine, and in Tyrrell's case there had been 22 fractures—scarcely any long bone had escaped.

Diagnosis of the idiopathic form can only be made by excluding those conditions causing rarefaction and atrophy of bone. A history of the disease in the relatives is important, and it must not be confounded with osteomalacia. As Fragilitas ossium may affect the fœtus *in utero*, and the fœtus be born prematurely with multiple fractures, the disease has a certain importance from the medico-legal standpoint.

Treatment.—Union is usually rapid and firm. We have no means of checking or curing the disease itself.

Frambœsia. *See* VENEREAL DISEASE (*Allied Diseases, Frambœsia*); YAWS.

Frame Food. *See* INFANT FEEDING (*Farinaceous Foods*).

Frangulin.—A glucoside, $C_{21}H_{20}O_9$, obtained from the bark of *Rhamnus frangula*; on hydrolysis it separates into *Emodin* ($C_{15}H_{10}O_5$) and the sugar *rhamnose* ($C_6H_{12}O_5$).

Fränkel's Pneumococcus. *See* EAR, ACUTE INFLAMMATION OF MIDDLE (*Causes, Micro-organisms*); EAR, MIDDLE, CHRONIC INFLAMMATION (*Bacteriology*); PNEUMONIA, BACTERIOLOGY OF (*Pneumococcus*); NOSE, ACCESSORY SINUSES, INFLAMMATION (*Bacteriology*).

Fränkel's Posture Test.—The reappearance of pus in the middle meatus of the nose after it has been all removed, and after the head has been held with the vertex dependent and the suspected side uppermost; it indicates antral disease. *See* NOSE, ACCESSORY SINUSES, INFLAMMATION (*Diagnosis of Chronic Suppuration in the Anterior Group of Sinuses, Fränkel's Posture Test*).

Frankincense.—Frankincense or *Thus Americanum* is an oleo-resin obtained from *Pinus palustris* and *P. tæda*; it is used as an antiseptic and slightly stimulant application for wounds and ulcers, and is contained in *Emplastrum Picis*; it has characters like turpentine. *See* RESIN.

Franzenbad. *See* BALNEOLOGY (*Austria, Alkaline*); MINERAL WATERS (*Alkaline*).

Franz Josef Water. *See* BALNEOLOGY (*Austria, Sulphated*).

Frauenhofer's Lines. *See* PHYSIOLOGY, BLOOD (*Pigment, Hæmoglobin*).

Freckles. *See* SKIN, PIGMENTARY AFFECTIONS OF (*Classification, Actinic*).

Free Martin.—The sterile cow-calf born co-twin with a bull-calf; it has been supposed to be not really a cow-calf but a pseudo-hermaphroditic bull-calf. The rule that the female twin born co-twin with a male is sterile does not apply to the human subject.

Freezing. *See* ANÆSTHESIA (*Local*); CRYOSCOPY; METEOROLOGY; TEMPERATURE, DEPRESSION OF; URINE, PATHOLOGICAL CHANGES IN (*General Properties, Freezing Point*).

"Frémissement."—A thrill. *See* ANEURYSM (*Signs and Symptoms, Thrill*); CHEST, CLINICAL INVESTIGATION OF (*Heart, Thrill*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Physical Examination, Palpation, Thrills*).

Fremitus. *See* CHEST, CLINICAL INVESTIGATION OF (*Palpation of Chest Wall*); THRILL; etc.

Frenkel's Exercises. *See* TABES DORSALIS (*Treatment, Massage and Gymnastics*).

Frenum.—Any slight band or bridle-like structure, such as the frenum or frenulum of the tongue, of the lips, of the clitoris, and of the prepuce.

Freyer's Operation.—Total extirpation of the prostate gland by the suprapubic route for the radical cure of prostatic enlargement.

Friars Balsam. Tinctura Benzoini Composita. *See* Benzoinum.

Friction. *See* Chest, Clinical Investigation of (*Auscultation*); Liver, Tropical Abscess (*Local Signs, Peritoneal Friction*); Mediastinum (*Chronic Mediastinitis, Diagnosis*); Pericardium, Diseases of (*Pericarditis, Physical Signs*); Pleura, Diseases of (*Acute Pleurisy, Physical Signs*).

Friedländer's Bacillus. *See* Ear, Middle, Acute Inflammation (*Causes*); Ear, Middle, Chronic Suppuration (*Bacteriology*); Scleroma Neonatorum (*Causes, Bacteria*).

Friedreich's Ataxia. — Hereditary ataxia. *See* Brain, Cerebellum, Diseases of (*Cerebellar Heredo-Ataxy, Diagnosis*); Tabes Dorsalis (*Diagnosis*).

Friedreich's Disease. — Paramyoclonus Multiplex. *See* Spasm (*Varieties, Paramyoclonus multiplex*).

Friedreich's Sign.—Diastolic collapse of the cervical veins in cases of adherent pericardium. *See* Pericardium, Diseases of (*Adherent Pericardium, Physical Signs*).

Friedrichshall.—A mineral water containing sulphate of magnesium and sodium salts. *See* Mineral Waters (*Sulphated, Bitter Waters*).

Frisch's Bacterium. *See* Nose, Examination of (*Secretion, Bacteria, Rhinoscleroma*).

Frontal Lobes. *See* Brain, Physiology (*Sensory Centres*); Physiology, Nervous System (*Cerebrum*).

Frontal Sinus. *See* Nose, Accessory Sinuses, Inflammation of (*Anatomy, Acute and Chronic Inflammation, Treatment*).

Frost. *See* Meteorology.

Frost-bite. *See* Ear, External, Diseases of (*Auricle, Frost-bite*); First Aid (*Sundry Accidents, Frost-bite*); Gangrene (*Varieties, From Cold, Frost-bite*); Raynaud's Disease (*Diagnosis*).

Fruits. *See* Diet (*Fruits, Composition*); Invalid Feeding; Sprue (*Fruit Cure*).

Fryer's Destructor.—A slow combustion furnace (without a forced draught) for the disposal of refuse; a fume cremator may be added.

Frying. *See* Invalid Feeding (*Preparation of Meats, with the Juices retained*).

Fuchsine.—A staining agent, consisting usually of a mixture of rosaniline and pararosaniline hydrochlorides, employed in microscopy; a cell readily stained with fuchsine is called a *fuchsinophile*; fuchsine has been used as a medicine in albuminuria.

Fucol.—An oily fluid obtained from various species of Fucus and Laminaria, recommended as a substitute for cod liver oil.

Fucus.—Seaweed. *Fucus dulcis* in certain parts of the world is used as food, and it has been employed as a remedy for obesity and as a diaphoretic and antipyretic. *Fucus vesiculosus* or sea-wrack contains sodium and iodine in considerable quantity, and has also been recommended in obesity and as "seaweed baths."

Fulgurant.—A descriptive term applied to the pains of tabes dorsalis; lightning-like in suddenness and severity.

Fuller's Earth.—A dusting powder, consisting of aluminium silicate. *See* Alum.

Fulmar Oil. — An oil obtained from several species of birds on the Island of St. Kilda, formerly regarded by some as the cause of the tetanus neonatorum once so common on the Island, but now known to have no such ill effects; it resembles cod liver oil.

Fulminating. — A term applied to diseases which develop very quickly, run a rapid course, and terminate fatally.

Fumaria.—Common Fumitory (*Fumaria Officinalis*) has been used as a tonic medicine; *Fumaria capreolata* (Earth-Smoke) has been employed for removing freckles; and *F. parviflora* has a reputation in the East in the treatment of malaria. Fumaria officinalis contains the alkaloid *fumarine* and fumaric acid ($C_4H_4O_4$).

Fumigation. *See* Disinfection; Syphilis (*Treatment of Secondary Syphilis, Mercurial Fumigation*).

Functional.—A term applied to non-organic diseases or disorders, to those not involving structural alterations. *See* Balneology (*Hysterical and Functional Paralysis*); Hemiplegia (*Hysterical or Functional*); Liver, Diseases of (*Functional*).

Fundus Oculi. *See* Retina and Optic Nerve (*Anatomy*); Tabes Dorsalis (*Symptomatology, Eye, State of Fundus*).

Three Cases of Furunculus Orientalis, or Aleppo Button.
(From the practice of Dr. Mackinnon of Damascus.)

Fundus Uteri. *See* Generation, Female Organs (*Uterus, Parts*); Puerperium, Physiology (*Height of Fundus*).

Fungi. *See* Diet (*Fungi, Lichens, and Algæ*); Toxicology (*Poisoning by Food Stuffs, Fungi*).

Fungus.—In descriptive Medicine the name fungus (mushroom) is given to any morbid growth of a spongy kind, such as a mass of exuberant granulations (*e.g.* fungus cerebri, f. testis, f. umbilicalis, etc.).

Fungus-Foot. *See* Mycetoma.

Funis.—The Umbilical Cord. *See* Fœtus and Ovum, Development of (*Umbilical Cord*); Labour, Accidental Complications (*Prolapsus Funis*).

Funnel Breast.—Funnel Breast (Germ. *Trichterbrust*) is that in which there is a marked depression over the lower part of the sternum; it has been ascribed to post-nasal adenoids, to tuberculosis, etc.

Furfuraceous.—A term applied to a branny form of desquamation of the cuticle (*furfur*, bran); scurfy; it is a well-marked character of such skin diseases as ichthyosis and psoriasis.

Furor.—Madness or mania; extreme excitement; *furor brevis* is a short-lived outburst of wild rage; *furor uterinus* is a synonym of nymphomania.

Furunculosis.—A condition in which multiple boils appear all over the body. *See* Boils and Carbuncle.

Furunculus.—A boil. *See* Boils and Carbuncle.

Furunculus Orientalis. Syn.: Oriental Boil or Sore.

Under this heading must be described an affection which, under various names, is exceedingly common in some parts of tropical countries. Sooner or later almost every one residing in the tropics suffers from boils, either singly or in crops, but in some places boils appear to be endemic as well as epidemic, and are well known under the local names Aleppo evil, Biskra button, Button de Crete, Dehli boil or sore, as also Garzebad, Jeypore, Lahore, Moultan, Meerut, Roorkee, Scinde, Umballa, Agra, and Aden boils or sores. Until very recently authorities differed as to the identity of these boils, but now almost all agree, and, for practical purposes at any rate, they may be said to be identical.

All ranks, sexes, ages, and classes of the population may be affected, and the exposed parts of the body are very prone to attack, although the boils may appear all over the body, except upon the palms of the hands and soles of the feet, and they are rare upon the scalp. Children are usually attacked on the face.

These boils are most frequently met with during the rainy season, and vary in number from one or two to fifteen or even more, although the smaller number is the most usual. Horses and dogs probably also suffer from this affection.

Debility certainly predisposes to boils, as also the combined effects of malarial influences, exposure to long-continued heat and residence in insanitary localities, aided perhaps occasionally by a scorbutic or syphilitic taint.

The *etiology* of this affection is in an unsatisfactory state. Lewis and Cunninghame thought that there was no evidence of any parasitic agency in the production of the disease, and considered that it was due to the chemical constituents of the water. Vandyke Carter considered that Delhi sore and Button de Biskra were identical, and thought it due to some fungus. Other observers think that the disease is due to inoculation by some insect.

If the boil be examined microscopically, infiltration of the skin and the areolar tissue, with lymphoid and epithelioid cells, is seen; these cells cluster round the blood-vessels and lymphatics. The nuclei of the cells are from 3 μ to 6 μ, the cells themselves from 7 μ to 9 μ. In 1885 Cunningham made a further investigation, and said that the diseased processes might be associated with and possibly caused by peculiar parasitic bodies which he found; they could be stained by gentian violet. His researches were confirmed by Frith, but Riehl subsequently found micrococci, as did Poncet, varying in size from 0·9 μ to 1·0 μ in the granulation cells. The whole matter is, however, at present indefinite.

[In 1903 Wright discovered a protozoon-like body in the tissues of the Dehli boil. This parasite, which is in all probability the cause of the condition, very closely resembles the Leishman-Donovan body found in the spleen and liver in kala-azar or tropical splenomegaly. Notwithstanding their apparent identity, the fact that they occur in two diseases so dissimilar as Dehli boil and kala-azar, suggests that they may be different, though allied organisms.]

The incubation of the disease varies from 3 to 16 days. After one attack a certain local immunity is enjoyed, and Bagdad Jews are said to have inoculated their children so as to prevent their disfigurement. The value of this practice is however doubtful, as the affection can be undoubtedly produced by inoculations upon persons who have previously suffered from it, but here again information is not absolutely accurate, and local rather than general immunity is most probable.

The *symptoms* of this affection are first itching, followed by the appearance of a reddish spot, which usually surrounds a hair follicle. A papule results, which gradually enlarges until a more or less appreciable tubercle, of a shotty feel, results. This in a few days softens, breaks down, and an ulcer is developed, which ulcer gradually spreads until an area of some two or three inches in diameter may result in severe cases. It is far more usually limited to an ulcer of perhaps an inch in diameter. The ulceration spreads by erosion; the margins are perpendicular, ragged, and surrounded by an areola. The surface of the ulcer discharges a sero-purulent matter, which forms scabs, as a rule covering the entire lesion. The onset of the disease is accompanied by more or less constitutional disturbance, malaise and fever being usually present and sometimes severe. The duration of the affection varies; sometimes the ulcer is small and heals rapidly, but it may last for a year. The duration is not much influenced by treatment in the present state of our knowledge.

With regard to *treatment*, great cleanliness is necessary to avoid auto-infection. The constitutional condition must be carefully inquired into, and tonics, such as iron, quinine, arsenic, and the mineral acids, are indicated. Change of air is very beneficial. In severe cases the patients should be sent either home or to some local sanatorium. The sufferers should be well supplied with nourishing food; stimulants may be required, and the bowels should be carefully regulated. Local treatment must depend upon circumstances. If the case is seen early, Dr. Fleming's treatment of applying strong mineral acid or potassa fusa to the pimple several times, so as to thoroughly destroy it and turn it into an ulcer, may be recommended, the ulcer being treated on general principles subsequently, but not irritated. Native practitioners often use the actual cautery in the early stage, and the writer has found this useful, as also Volkmann's spoon. If the sores are small in extent they may be covered by iodoform collodion. If the pain is severe, as it sometimes is, belladonna ointment is indicated, or a compress of a 10 per cent solution of chloral hydrate in glycerine and water. It is well to remember that after healing these boils frequently leave a scar, which, if on the face, may be unsightly. Patients should be warned of this; sometimes a scar may take the form of a brown mark; hence the name "date mark" given in some regions to the affection.

Fuscin. *See* Pigments of the Body and Excreta (*Melanins*).

Gadara. *See* Balneology (*Palestine, Thermal Waters*).

Gadflies. *See* Myiasis (*Insect Stings and Bites*); Stinging Insects (*Gnats, etc.*).

Gadic Acid.—A fatty acid ($C_{28}H_{58}O_4$), contained in cod-liver oil. *See* Cod-Liver Oil.

Gaduol.—A preparation said to be an extract of cod-liver oil, and recommended in the bronchitis of children, etc.

Gag. *See* Palate (*Cleft Palate, Operation, Instruments*).

Gait. *See* Alcoholism (*Intoxication*); Brain, Inflammations (*Acute Encephalitis, Clinical Features*); Brain, Cerebellum (*Hæmorrhage, Reeling Gait*); Chorea (*Symptoms, Gait*); Deformities (*Coxa Vara, Symptoms*); Muscles, Diseases of (*Pseudohypertrophic Paralysis, Symptoms*); Nerves, Multiple Peripheral Neuritis (*Symptoms, Gait*); Osteomalacia; Paralysis (*Spastic Paralysis, Clinical Aspect*); Paralysis (*Paralysis Agitans*); Senile Insanity (*Old Age, Gait in*); Tabes Dorsalis (*Symptomatology, Disturbance of Gait*).

Galactagogue.—A medicine increasing or stimulating the flow of milk, *e.g.* jaborandi. For nursing women, milk is itself the best galactogogue.

Galactocele. *See* Mammary Gland, Diseases of (*Cysts, Milk-Cyst*); Puerperium, Pathology (*Affections of the Mamma, Galactocele*).

Galactorrhœa.—Persistent secretion of milk; sometimes used as synonymous with polygalactia.

Galactose.—A carbohydrate ($C_6H_{12}O_6$) formed from lactose (milk-sugar) by boiling with a dilute acid, glucose being formed at the same time. *See* Liver, Physiology of (*Regulation of Supply of Sugar to the Body*); Physiology, Food and Digestion (*Carbohydrates, Galactose*).

Galbanum.—A gum resin, obtained from one of the Umbelliferæ (*Ferula Galbaniflua*), with an aromatic odour and a bitter taste, and containing a volatile oil, a resin, a gum, and umbelliferone; there is no official Emplastrum in the British Pharmacopœia, although there is one in that of the United States; internally the medicine is given in doses of from 5 to 15 grains, or as the official *Pilula Galbani Composita* (dose, 4 to 8 grains), which contains also asafetida; it is now little used.

Galea.—A headache; also, a bandage for the head; also, the caul.

Galeanthropy.—That form of insanity in which the patient believes himself to have been transformed into a cat.

Galen, Veins of. *See* Brain, Physiology of (*Venous Circulation*); Meninges of the Cerebrum (*Anatomy and Physiology, Causes of Hydrocephalus*).

Galenism.—The humoral theory of Medicine as maintained by Galen (b. 131, d. 201 (*circa*)). *See* Medicine, History of.

Galipine.—An alkaloid found in Angostura bark, the bark of *Galipea cusparia* ($C_{20}H_{21}NO_3$).

Gall, Ox. *See* Fel Bovinum Purificatum.

Galla. *See* Galls.

Gall-Bladder and Bile Ducts, including Gall-Stones, Diseases of.

See also Abdomen, Injuries of (*Lesions of Gall-Bladder*); Abdominal Tumours, Diagnosis of (*Gall-Bladder*); Appendix Vermiformis, Appendicitis (*Diagnosis*); Ascites (*Distinction*); Digestion and Metabolism (*Bile, Gall-Stones*); Hydatid Disease (*Diagnosis*); Intestines, Diseases of (*Anatomy, Gall-Bladder*); Intestines, Surgical Affections of (*Obstruction, Causes*); Kidney, Surgical Affections of (*Hydronephrosis, Diagnosis*); Liver, Diseases of (*Anatomical Abnormalities of, Floating Lobe*); Liver, Tropical Abscess (*Diagnosis*); Malaria (*Diagnosis from Gall-Stones*); Ovaries, Diseases of (*Cysts, Diagnosis*); Pancreas, Diseases of (*Pancreatitis and Gall-Stones*); Physiology, Food and Digestion (*Large Intestine, Liver, Bile*); Pigments of the Body and Excreta (*Bile Pigments*); Stomach, Surgical (*Gastric Ulcer, Complications, Gastric Fistula*); Visceral Pain (*Gall-Ducts*).

Anatomy.—The gall-bladder is a pear-shaped sac, measuring three and a half inches in length and two inches at its greatest breadth, which lies against the under surface of the right lobe of the liver, filling up a slight fossa to the right of the quadrate lobe and projecting into the peritoneal cavity.

Its fundus, when the gall-bladder is distended, normally projects slightly beyond the free margin of the liver immediately under the ninth costal cartilage, and from this position the cyst passes backwards and slightly to the left, gradually narrowing until its neck ends in a sigmoid curve, the terminal portion of which turns downwards to its junction with the cystic duct near the right extremity of the transverse fissure of the liver. Its upper surface, as a rule, is attached to the under surface of the liver by connective tissue, the peritoneum covering the fundus and lower surface, and passing thence to the liver; but occasionally it is completely invested by peritoneum. At times also a mesentery is formed on the under surface by an extension outwards of the free border of the lesser omentum to the fundus.

The cystic duct passes backward, slightly downward, and to the left for rather less than one and a half inches, and joins the hepatic duct at an acute angle to form the common bile duct. The hepatic duct measures about two inches in length, and is formed by the junction at a very obtuse angle of a branch from the left, and another from the right lobe of the liver, which issue at the transverse fissure. Thence it descends somewhat to the right within the gastro-hepatic omentum, lying in front of the portal vein, and having the hepatic artery to its left.

The common duct averages three inches in length and passes backward and downward between the layers of the gastro-hepatic omentum, having the same relation to the portal vein and hepatic artery as the hepatic duct. Passing behind the first part of the duodenum it continues downward on the inner and posterior surface of the second part in intimate relation with the head of the pancreas, through which it runs in about 60 per cent of cases. For a short distance it is in contact with the right side of the duct of Wirsung. Along with this duct it perforates the muscular coat of the duodenum, runs obliquely for three-quarters of an inch in the submucosa, and then opens into the lumen of the bowel about three inches from the pylorus by an orifice common to the two ducts known as the ampulla of Vater. In some cases the ducts open separately, in others the pancreatic duct opens into the common bile duct (6).

The cystic duct normally admits a No. 5, the common duct a No. 7 catheter; but at different parts they both vary in size, the cystic being narrowest at its termination, while the common duct shows two dilatations, one just at its beginning and another near its termination. At its entrance into the duodenum it is so narrow that it only admits a fine probe.

The ducts have each three coats,—serous, muscular, and mucous,—the last being studded with mucus-secreting glands. In the cystic duct the mucous membrane is thrown into folds, which, especially at its termination, act somewhat as a valve.

The gall-bladder, except where in contact with the liver, has the same three coats, and its mucous membrane also is abundantly supplied with glands which secrete mucus. Its normal capacity is about six drachms, but under varying pathological conditions its size undergoes great alterations.

The gall-bladder gets a free supply of blood from a branch of the hepatic artery—the cystic artery—which in turn sends a branch along the cystic duct to anastomose with an offset from the gastro-duodenal artery. The veins empty themselves into the portal vein, and the lymphatics drain into glands which lie in the free border of the lesser omentum.

Accompanying the arteries are branches from the cœliac plexus of the sympathetic.

The lower surface of the gall-bladder is in relation with the hepatic flexure of the colon, and, toward its neck, with the first part of the duodenum and occasionally the pylorus. The tip of the fundus just touches the anterior abdominal wall, and occasionally the right border of the great omentum lies in contact with it. In pathological conditions, however, there may be great alterations in the relations of the parts.

Congenital Malformations. — The gall-bladder is sometimes partly bifid, or by a central constriction it may assume an hour-glass shape. Occasionally it and even the larger bile ducts are absent or represented merely by fibrous cords.

A malformation of the liver not infrequently met with is of great importance in gall-bladder work. It consists of a tongue-shaped prolongation of the right lobe, which may project below the costal margin for several inches and carry the gall-bladder down with it, though it is more common for the cyst to be situated just to the left of the abnormal (Riedel's) lobe.

Congenital Obliteration of the Bile Ducts is sufficiently common to merit special attention. The exact cause of this condition has not been definitely determined, but it apparently consists of an inflammatory process affecting the bile ducts during intra-uterine life, tending to be followed by complete obliteration of their lumen and associated with some degree of cirrhosis of the liver.

It is probable that the cases which have been reported from time to time under the title "absence of the gall-bladder and bile ducts" should really be relegated to this category, as it is not unusual for some part of the extra-hepatic biliary apparatus to be absent in this disease. Where the patient has lived some time it is usual to find more or less marked biliary cirrhosis, and the liver occasionally, and the spleen usually, are somewhat enlarged.

In a few cases there has been reason to suspect the presence of congenital syphilis; but in the majority the parents have apparently been in perfect health.

The male sex suffers more frequently than the female, and in several recorded instances several members of a family have been affected by the disease.

That the process is a local inflammatory disturbance is evident from the fact that at birth the patient has usually been of normal weight, and seemed normal in all respects.

As a rule the first symptom pointing to any serious condition is the progressive deepening of the jaundice which is either present at birth or soon supervenes. An early symptom in the disease is the occurrence of hæmorrhages from various parts—from the umbilicus after separation of the cord, from the nose, stomach, or intestine, or into the subcutaneous tissues. Where the affection has started early in intra-uterine life the fæces will from birth be pale, but if toward the end of the period of gestation, normal meconium will first be extruded, and this will be followed by clay-coloured stools. Unless death results from hæmorrhage, the general nutrition suffers considerably after a time; but, if not carried off by some intercurrent disease, the patient may live on for months before succumbing to the effects of the continued malnutrition.

The only condition with which this disease is likely to be confounded is icterus neonatorum, and in the early stages it is impossible to distinguish between them; but the continuance of the jaundice and the supervention of serious sequelæ soon render a diagnosis easy.

The prognosis is uniformly bad; even in those cases where there is merely narrowing of the ducts and inspissation of the bile, life has never been prolonged more than eight months.

So far no treatment has seemed to do any good; but since the disease is apparently a local inflammation, the duodenum should be rendered as aseptic as possible by the administration of grey powder, and the bile be kept as thin as possible by giving as much fluid as can comfortably be ingested, in the hope that if the disease has not progressed to obliteration of the ducts the process may stop.

Injuries of the Gall-Bladder or Bile Ducts are not common, but several have been reported. The symptoms vary considerably, according to

the condition of the bile, which may or may not be pathological. Practically invariably in cholelithiasis, or in any inflammatory condition affecting the bile channels, the contents of the gall-bladder and ducts contain pyogenic microbes, and if, in the presence of these, the ducts or gall-bladder are so injured as to lead to extravasation, an acute septic peritonitis is set up, which, unless promptly treated, is very apt to end fatally. Adequate surgical treatment carried out shortly after rupture, has on several occasions yielded good results. Where, however, the bile is normal, no micro-organisms are present, and, even if extravasation takes place, there is less risk of immediate septic peritonitis; though, if the extravasated fluid be not removed, peritonitis will certainly supervene sooner or later.

Many of the recorded cases of rupture of the gall-bladder have occurred from comparatively slight sudden pressure, induced by straining at stool, by vomiting, by sneezing, or by blows over the hepatic region; but in these cases, in all probability, there has been predisposition to rupture in the shape of thinning from ulceration or long-continued distension. The course and symptoms in such a case will be those of perforation of the gall-bladder, a subject which will be considered below.

In non-pathological conditions of the gall-bladder and bile ducts the injuries, in the reported cases, have taken the shape of gunshot wounds, of perforating abdominal wounds by sharp instruments, or of the passage of a heavy conveyance over the hepatic area. In cases where a perforating abdominal wound is present operation would probably be done as soon as possible, and before any symptoms pointing directly to injury to the bile channels had arisen. On the true nature of the condition being determined the extravasated bile should be mopped out by sponges, the rent in the viscus closed by Lembert sutures, and drainage of the right kidney pouch established. If the patient does not die from the shock of the injury, and operation is undertaken sufficiently early, the chances of recovery from such a condition should be very good.

Where the injury has resulted from the passage of a heavy body over the abdomen, there has not in uncomplicated cases been much evidence at first of any serious visceral lesion.

In a few days after the accident, jaundice will appear along with symptoms of peritonitis, which may become general, and though not at the outset very acute, it ultimately becomes so. Examination of the abdomen will give evidence of the presence of free fluid in the peritoneal cavity, and the stools will be clay-coloured or paler than normal.

Whenever it is clear that there has been escape of bile into the peritoneal cavity the abdomen should be opened, the free fluid mopped out by sponges if localised, or flushed out by sterile normal salt solution if free in the general cavity of the peritoneum, and the wound closed if possible, and drainage of the abdomen established.

If this course is pursued the prognosis is not hopeless. In one case recovery took place, although the operation was not undertaken until thirty days after the injury to the bile channels.

Cholelithiasis

Though gall-stones are extremely frequent, various estimates giving them as occurring in from 4 to 12 per cent of all autopsies, yet it is only in a minority that any distinct clinical evidence of their presence is afforded.

In number and size the gall-stones found vary greatly in different cases. Occasionally a single stone is found, while as many as 7802 have been obtained in one case; they may be so small as just to be perceptible, while, on the other hand, one has been described 5 inches by $1\frac{3}{4}$ in.

As a rule a single stone is rounded or pyriform, but when many are present they are more usually small and faceted. In colour they vary from almost pure white to dark olive green, according to the relative amount of bile pigment present. As might be supposed from their being composed almost entirely of cholesterin, they are at times sufficiently soft to be readily crushed between the finger and thumb, but on the other hand they may be firm and hard. Their specific gravity is just above that of water.

The cholesterin, which is the main constituent in a biliary calculus, is derived, not as used to be thought, from the bile, but from the epithelial lining of the gall-bladder or bile ducts; indeed, the excretion from mucous membranes generally, especially if inflamed, contains quite as large a proportion of cholesterin as does the bile.

It seems probable, therefore, that the origin of gall-stones is to be sought, not in any alteration in the characters or rate of flow of the bile, but rather in a local condition of the mucous membrane of the bile passages. During the last twenty years from all sides evidence has been accumulating which tends to show that this preliminary condition consists of a bacterial infection of the bile channels from the intestine. Normally bile is sterile, but in practically all cases of cholelithiasis micro-organisms can be obtained if carefully searched for. As a rule the bacillus coli communis is the microbe found, but the typhoid bacillus, staphylococci, and streptococci have also been found, and it is probable that other bacteria may induce a cholangitis sufficient to give rise to a hypersecretion of cholesterin.

More recently gall-stones have actually been experimentally produced by inserting into the

gall-bladder various species of bacteria specially attenuated by cultivation in media containing increasing proportions of bile. It may, therefore, for practical purposes, be taken as proved that gall-stones owe their origin to a bacterial invasion of the bile channels, giving rise to a catarrh of the mucous membrane and a deposition of that portion of the cholesterin secreted which is in excess of what the bile salts are able to hold in solution. The mere presence of this deposit will, of course, keep up the catarrh even should no further microbic infection take place.

While this is so it is necessary that we should consider several conditions which seem to predispose to cholelithiasis.

Of these the most important is the character of the food. There seems to be little doubt that the ingestion of excess of carbohydrates as compared with nitrogenous food is a fairly common precursor of the onset of gall-stones. While this may be due to the fact that, with a diminution in the amount of albuminous material consumed, there is a corresponding deficiency in the bile salts which dissolve cholesterin, it seems more probable that it is associated with an increased tendency to catarrh of the stomach and duodenum from a greater liability to abnormal fermentation in the stomach. Normal gastric juice seems to kill most disease-producing bacteria, and it seems probable that any alteration of the contents of the stomach and duodenum would tend to increase the risk of the introduction of microbes from the bowel into the common duct.

Gall-stones may occur at any age from infancy upward, but they are distinctly more prevalent in the later decades of life. Why this should be so is not on the surface quite apparent; but it has been suggested that the reason for biliary calculi being found more frequently in autopsies on elderly subjects is that the walls of the bile passages undergo great atrophy in old age, and are consequently unable to expel any small concretions which form.

Sedentary habits, from whatever cause arising, appear undoubtedly to predispose to cholelithiasis. Lack of sufficient exercise, besides affecting prejudicially the general tone of the tissues, is not infrequently associated with dyspepsia and gastro-intestinal disturbance, and it is possible this is the explanation of the coincidence. It is probable, also, that defective respiratory movement and absence of contraction of the abdominal muscles tend to help toward more or less stagnation of the bile in the gall-bladder and bile passages. It has been found that if the bacillus coli be injected into the gall-bladder of a dog after the common duct has been ligatured, an acute cholecystitis develops, whereas if the ducts are free no bad symptoms appear. Similarly the stagnation of bile consequent on deficient exercise may tend toward the more ready origin and propagation of a catarrh of the ducts.

Gall-stones occur much more frequently in women than in men—in the proportion of about 3·5 to 1. This is probably associated with their taking less exercise, but it is possible that the habit of wearing corsets, which depress the fundus of the gall-bladder, and so impede the flow of bile from it, helps as an etiological factor.

Symptoms.—The symptoms caused by gall-stones vary greatly in different cases. It is probable that this is in the main due to the locality in which they are situated and to the presence or absence of complications.

In this section only uncomplicated cholelithiasis will be considered, the complications being relegated to a later chapter—a method which, though pathologically somewhat illogical, is clinically convenient.

(*a*) *Gall-stones in the Gall-bladder.*—Though most frequently found in the gall-bladder, it is a mistake to suppose that all biliary calculi originate there. So long as they remain in the gall-bladder it is probable that they give rise to no special symptoms, except possibly some sense of slight uneasiness and discomfort in the epigastrium, and some tenderness below the right costal margin, unless the inflammatory condition, which gave rise to their formation and which is perpetuated by their presence, has spread to the peritoneal covering of the cyst.

(*b*) *Gall-stones in the Cystic Duct.*—When a calculus reaches the cystic duct there is sooner or later some symptom of its presence. A typical attack of gall-stone colic, when the stone is lodged in or extruded into the cystic duct, consists of very severe pain beginning under the right costal margin, and radiating thence toward the epigastrium and umbilicus, and round the right side, usually toward the subscapular region, but occasionally even to the right shoulder. The onset of pain is often apparently without any determining factor, though in some patients it will be found usually to occur after taking food. Its duration varies very considerably in different patients, and at different times in the same patient. Not infrequently it ends with the onset of vomiting. In a severe seizure there is very great collapse, so that the condition may closely simulate angina pectoris, perforation of a gastric ulcer, or other grave abdominal catastrophe, and death has on more than one occasion resulted from the severity of the pain.

The vomiting may persist after the pain has quite ceased, and the stomach may continue irritable for days after, so that the ingestion even of liquid food brings on another attack of vomiting, and resort has to be had to rectal alimentation. If the common duct is free, some bile will usually be ejected after the contents of the stomach have come away.

Associated with this seizure there is a greater or less degree of local peritonitis giving rise to rigidity of the upper segment of the right rectus muscle and tenderness over the gall-bladder region, particularly at a point about midway between the ninth costal cartilage and the umbilicus.

During such an attack there is frequently some distension of the gall-bladder, but this can rarely be made out on account of the muscular rigidity and the excessive tenderness.

Jaundice is not usually an accompaniment of gall-stone colic in these cases, though it may appear at the end of twenty-four hours and persist as slight icterus for two or three days. In such a case it is due to catarrh spreading to the common or hepatic duct, and interfering with the flow of bile. It is not common to find a stone in the motions after such an attack as that described above, but one or more may be found.

It is important to remember that neither the absence of calculi nor a failure in the appearance of jaundice negatives the diagnosis of cholelithiasis.

While the description given above applies to a typical cholelithic attack, it must not be supposed that in every case the symptoms will be so definite or so severe; but even the milder seizures, so-called "spasms," will be found to conform more or less, and to differ only in the intensity of the individual symptoms.

They may be repeated at intervals of a few days, or months may intervene between one attack of colic and another. In the intervals the patient may be quite comfortable, but local examination will usually detect a point between the costal margin and the umbilicus where deep pressure will elicit tenderness, and if a gall-stone be impacted in the cystic duct a tumour may be felt, due to the gall-bladder being distended with mucus.

(*c*) *Gall-stone in the Common Duct.*—The symptoms of gall-stone colic, when the calculus is in the common duct, resemble in many ways those following on the impaction of a stone in the cystic duct, but differ in respect to the seat and distribution of pain and to the occurrence of jaundice.

The pain in such a case begins in the epigastrium, and though usually extending to the right scapula may radiate to the left side of the abdomen and through to the inter-scapular space. The tenderness will also be found most marked in the middle line about midway between the umbilicus and ensiform cartilage.

Jaundice is usually present, but varies in intensity according as the stone does or does not completely block the duct, or become impacted at its entrance into the duodenum. Where the stone is too small quite to fill the lumen of the tube the jaundice will resemble that found in the last class, but its onset will be earlier, and it will last longer. Here, the immediate cause of the icterus is the obstruction due to the calculus, as well as that due to the inflammatory swelling of the mucous membrane. Should the stone quite fill the duct, or should it become impacted at the entrance to the bowel, the jaundice will be persistent, and may become extreme. In that event there will be much greater chance of rapid deterioration of health, and greater difficulty in diagnosing the condition from malignant disease. There will also be the usual signs and symptoms of deep jaundice, and if long persisting especially, what from a surgical point of view is of great importance, a marked tendency to hæmorrhage.

As was found to be the case when a stone was present in the cystic duct, so here, between the seizures, if the jaundice is not persistent, the patient may be fairly well, though interference with the general health is much more frequent than in the former case. Tenderness will always, or nearly always, be elicited if deep pressure be made midway between the umbilicus and the tip of the sternum, and the right rectus will usually be found rigid.

In many cases infective cholangitis supervenes, a condition which will be described later, and not infrequently the infective condition spreads to the pancreas, producing a pancreatic catarrh or a chronic, subacute, or even an acute pancreatitis (7) and (8).

Diagnosis.—In uncomplicated cholelithiasis the diagnosis will not as a rule present much difficulty. The situation, character, and distribution of the pain and tenderness along with the accompanying symptoms will usually be sufficient to render the case clear. The locality in which the stone is situated can, for the most part, be determined by noting the point of greatest tenderness. When this is in the middle line the calculus will probably be in the common duct, and the pain in the back will be more central; whereas if the stone is in the cystic duct the point of greatest tenderness will be somewhere along a line between the umbilicus and the ninth right costal cartilage, and the pain will be felt to radiate to the right subscapular region. Should the pylorus, however, be adherent to the cystic duct at the site of the stone, the pain and tenderness may radiate also to the left scapular region, and in such a case there will usually be some degree of dilatation of the stomach or other gastric symptoms pointing to this complication.

If the common duct is quite blocked, jaundice will become intense, and there may be great difficulty in eliminating malignant disease as a probable cause of the illness. The previous history of attacks of "spasms," and particularly the account of severe pain at the onset of the illness, together with local tenderness, will be important indications tending to show that the disease is simple. On the other hand, in malignant disease of the head of the pancreas

or in the bile ducts, severe pain is not a common symptom, though it does occur, and the wasting and deterioration of the general health are more rapid than in jaundice due to impacted stone. Where the cause of the disease is simple it is distinctly unusual to find a distended gall-bladder; but in obstructive jaundice dependent on malignant disease, especially cancer of the head of the pancreas, this condition is the rule. It should, however, be remembered that gall-stones are probably invariably present before primary malignant disease of the gall-bladder or bile ducts, and in any particular case both conditions may be present. The absence of pain at the onset of the last attack of persistent jaundice raises the suspicion of cancer, and in such a case may be so striking as to lead the patient himself to remark on the fact.

Should a positive diagnosis be impossible, an exploratory incision will clear it up, but great help may be derived in such a case by a careful examination of the urine and fæces, since in common duct cholelithiasis of some standing it is usual to find associated pancreatic inflammation, when pancreatic crystals can be found in the urine and excess of neutral fat in the motions (7) and (8).

Aspiration of a distended gall-bladder or sounding for gall-stones should never be resorted to, as neither gives any information which cannot be obtained by a careful examination of the patient, and both operations are dangerous.

Treatment.—Once gall-stones have formed, it is probable that no means at present at our disposal suffice to dissipate them; but as some medical authorities hold a contrary opinion it will be well to consider here the various remedies which are supposed to have this power.

The drug which is most in favour at the present time is olive oil. It should be given in large quantity, as much as half a pint per day being recommended by some. There can be no doubt that if the oil could reach the calculi in the gall-bladder or bile ducts, and remain in contact with them, it would bring them into solution, as olive oil has been found to dissolve 68 per cent of a gall-stone placed in it for two days. Oleic acid has a similar but rather more rapid effect, and a solution of animal fat also has a tendency to soften biliary concretions. There is, however, no evidence to show that ingested oil can come into direct contact with calculi in the bile passages. It has been suggested that the good effect said to be obtained may be explicable on the supposition that an increased absorption of fat in the form of fatty acids and soaps leads to a greater proportion of these in the bile, and that they are the active ingredients in causing the dissipation of the concretions.

It seems, therefore, just possible that the administration of oil may in some cases, especially where the concretions are small, lead to their dissolution, or so reduce their bulk as to permit them to pass: but it should be remembered that excess of fat in any form is apt to induce dyspepsia and catarrh of the stomach and duodenum, and in this way may tend to aggravate the condition which it is sought to relieve.

So far as my own experience goes, I cannot say that in any case, even after treatment by this means continued over a lengthened period, I have seen any such material benefit as other writers describe.

The so-called saline cholagogues do not affect gall-stones introduced into solutions containing them, so that their administration is not likely to have much effect in ridding a patient of calculi once they are formed, though they may relieve the associated catarrh.

Chloroform, ether, turpentine, and several other substances readily dissolve cholesterin; but there is no evidence to show that, when taken by the mouth, they are excreted in the bile in such quantity as to have any effect in removing gall-stones in the gall-bladder or bile ducts.

During an attack of cholelithic colic the pain is so severe that not infrequently it is necessary to give morphine; but in other cases, where the pain is less intense, it may be relieved by the administration of a tumblerful of water as hot as it can comfortably be taken and the local application of hot compresses.

Surgical Treatment.—(*a*) When gall-stones in the gall-bladder or cystic duct give rise to symptoms, though urgent need for operative interference is less than in those cases in which the common duct is occupied by a calculus, yet in a certain number operation will be called for, notwithstanding the absence of those complications, inflammatory and other, which will be discussed later.

In simple cholelithiasis the two conditions which would seem to indicate necessity for surgical interference are gall-stone colic, recurring so frequently as to interfere with the general health of the patient or prevent him filling his position in society, and enlargement of the gall-bladder without jaundice, even in the absence of pain.

It should also be remembered that delay in resorting to operation means increased risk, from shrinkage of the gall-bladder and the formation of adhesions, should it become necessary to operate later; while there seems little doubt that the continuous irritation from the presence of gall-stones predisposes to malignant disease of the gall-bladder and cystic duct. In addition there is always the risk of acute inflammatory disturbance supervening, and operation having to be done with the patient in a bad condition to withstand surgical interference.

Cholecystotomy.—The operation most generally useful is that of cholecystotomy. A vertical incision through the outer border of the right

rectus is that which I prefer, but some surgeons habitually use an incision through the right semilunar line, while Kocher has advised, and many surgeons employ, an oblique incision parallel to the right costal margin. If, on opening the abdomen, the gall-bladder be found distended, it should be aspirated, and then opened, after the parts have been isolated by sterilised gauze. A pair of forceps or a small scoop should then be introduced, and any gall-stones present in the gall-bladder removed. After the gall-bladder has been cleared, the fingers are passed along the outside of the ducts (any adhesions which may have been present having previously been broken down), and the cystic duct is searched for calculi. If any be found they should be manipulated backward into the gall-bladder and thence removed. Sometimes it is necessary to crush a stone in the duct before it is possible to return it into the gall-bladder; at other times the calculus is so large or so hard that this manœuvre is impossible, and then it becomes necessary to incise the duct in order to remove the concretion. Should this latter course be adopted, the incision in the duct should be carefully stitched up, a continuous catgut suture being used for the mucous membrane, and a continuous silk suture for the peritoneal investment.

The gall-bladder and cystic duct having thus been cleared, a non-perforated thick walled rubber tube should be inserted into the opening in the gall-bladder, and the edges of the incision in the gall-bladder should be caught up by a purse-string suture, which when tightened around the tube prevents any leakage; the drain is also fixed in position by a fine seven-day catgut stitch, which dissolves when the tube is ready to come away. One or two similar sutures fix the gall-bladder to the abdominal aponeurosis. If there has been much cholecystitis, drainage should be maintained for a longer period.

Even when it is not possible to bring the gall-bladder up to the anterior abdominal wall the tube should be inserted into it, and the purse-string suture applied round the margin of the incision in the gall-bladder, so that when tightened it will draw the edges of the incision closely round the tube and prevent any escape of bile into the peritoneal cavity. This plan is, I consider, much better than that of stitching up the incision in the gall-bladder, since it is of the utmost importance that, if recurrence of cholelithiasis is to be prevented, the catarrhal condition of the mucous membrane should be removed by the employment of free drainage.

The results of cholecystotomy in uncomplicated cases has been in my practice less than 1 per cent, and the after-histories of the cases have been most satisfactory and free from recurrence (11).

(*b*) In certain cases cholecystectomy or complete removal of the gall-bladder is advisable, as for instance when the gall-bladder is atrophied and useless, when the cystic duct is ulcerated or strictured, when the gall-bladder is thickened by chronic inflammation, or is the seat of phlegmonous inflammation, or gangrene, or if there is a suspicion of malignant disease. The operation is rendered more easy by placing a sand-bag under the region of the liver or by the use of a special table.

The mortality is only a little more than that of cholecystotomy, barely 2 per cent (9).

(*c*) When the gall-stones are in the common duct, operation is imperative.

Choledochotomy.—The operation of election is choledochotomy, which is rendered comparatively easy by the position just mentioned and by an upward extension of the incision (10). The duct may be opened and the stone extracted, either by direct incision through the wall of the canal at the site of the calculus, or by incising the second part of the duodenum and stretching the papilla, or slitting up that part of the duct which runs in the posterior wall of the duodenum. The duct, or bowel, should then be carefully sutured, and the gall-bladder or duct drained in the manner already described. Separate drainage of the peritoneal cavity may be advisable if the parts have been soiled, but not otherwise.

Cholecystenterostomy.—In a few cases after choledochotomy, especially if there be interstitial pancreatitis, it may be advisable to perform cholecystenterostomy, *i.e.* to make a direct artificial communication between the gall-bladder and duodenum. When the gall-bladder is distended, this is a very simple matter; but as a rule, in these cases, the gall-bladder is contracted, and the operation then is one of some difficulty. In my experience the junction has been most expeditiously effected by the aid of a Murphy button or by suture over a decalcified bone bobbin.

In all cases of cholelithiasis, after recovery from operation, the patient should be instructed to attend to certain details of after-treatment. The bowels should be kept regular, preferably by the use of mild salines, of which probably the natural Carlsbad water is the best. The diet should be so regulated as to obviate the likelihood of dyspepsia, and, for this reason, the patient should be advised to avoid over-indulgence in sweet or starchy foods, or highly seasoned dishes. A sufficiency of albuminous food should be taken, either in the shape of meat or fish, or of vegetable food-stuffs which contain a large proportion of nitrogen. Alcohol should be avoided except at meals, and then should be well diluted.

A sufficient amount of exercise should be taken daily, as thereby the general nutrition will be improved, and the likelihood of fermentative changes taking place in the contents of the stomach and duodenum diminished.

Cholecystitis

Simple catarrh of the gall-bladder, like that of

the bile ducts, may be either acute or chronic. As the acute affection is always associated with a similar condition in the ducts, and as it is the latter that gives rise to the most evident symptoms, the consideration of the former will be deferred until the latter is discussed.

A chronic catarrh, as has already been pointed out, is probably an invariable precursor of the formation of gall-stone in the gall-bladder, but, as a rule, this condition gives rise to few symptoms other than evidences of dyspepsia, until calculi have formed.

The form of chronic catarrh which does give rise to symptoms is that usually following on the presence of gall-stones and persisting after these have disappeared, but occasionally originating as the sequel to an acute cholecystitis, like that sometimes present in association with typhoid fever, influenza, and other acute affections.

In these cases the local condition depends in great measure on the duration of symptoms. Where these have lasted only for a short time the gall-bladder may be somewhat dilated, though it is rare to find it so large as to be palpable through the abdominal wall; on the other hand, if the patient has been suffering for a long period the walls of the gall-bladder tend to become thickened, and the gall-bladder itself tends to contract. Adhesions to the neighbouring viscera may or may not be present according as the inflammatory disturbance has or has not extended to the peritoneal coat. As a rule, however, if there has been well-marked and repeated gall-stone colic, or if the preceding cholecystitis has been acute and associated with marked tenderness, there will be more or less adhesion present. The gall-bladder will usually be found to contain thick, ropy mucus, sometimes so inspissated as to resemble grains of boiled sago; but it may be so contracted that the cavity is practically obliterated,—a form to which the term *cholecystitis obliterans* may be properly applied.

The symptoms in this condition simulate very closely those due to cholelithiasis, but the colic is for the most part less severe, and the tenderness is either absent or much less marked. Jaundice is almost invariably absent, but, rarely, it does occur. In one case I treated recently the attacks of colic were invariably preceded by slight jaundice; in this case it seemed as if the thickened mucus was allowed to pass until a catarrhal condition of the mucous membrane in the common duct so lessened the calibre of the passage as to retard the flow of the mucus and thus set up painful spasm.

Medical treatment similar to that advised for acute catarrh will, if persisted with, get rid of symptoms in most cases; but in some, especially where there are adhesions, the gall-bladder will require to be opened and drained until the inspissated mucus disappears from the discharge. At the same time any adhesions which are present should be broken down. If the cavity of the gall-bladder be almost obliterated, the best mode of treatment will be to perform cholecystectomy.

Empyema of the Gall-bladder.—Normally bile removed from the gall-bladder will be found sterile, but experimentally it has been shown that if the outflow of bile and mucus from the gall-bladder be obstructed pyogenic micro-organisms make their appearance.

Pure cultures of staphylococci, streptococci, and bacillus coli communis have been introduced into the gall-bladder, where there was no obstruction to the outflow, without producing any untoward symptom. This experimental evidence is supported by clinical experience, since in every case in which empyema of the gall-bladder occurs it will be found that there has been present some cause of obstruction in the cystic duct; either a gall-stone has become impacted in the duct, or the lumen has become diminished as a result of a chronic catarrh, of kinking from adhesions, or of malignant disease.

In the great majority of cases the onset of empyema is preceded by cholelithiasis, though it may occur as a consequence of obstruction of the cystic duct from any cause—chronic catarrh, cancer of the duct, hydatids, etc.

From whatever cause arising, the onset of suppuration is followed by dilatation of the gall-bladder and localised peritonitis in its neighbourhood. Should the case be allowed to progress, the gall-bladder will either rupture into the general peritoneal cavity, giving rise to peritonitis, or it will contract adhesions to the neighbouring hollow viscera or the parietes, and relief may occur by the discharge of pus either into the intestinal canal or through the skin.

The symptoms leading up to empyema will, of course, vary with the cause, but mostly there will be the ordinary symptoms of cholelithiasis. With the supervention of suppuration there will appear a swelling under the right costal margin. The tumour will be found to be somewhat pear-shaped, and will be directed along a line reaching from the tip of the ninth costal cartilage to a point in the middle line about an inch below the umbilicus. It will move with respiration in the earlier stages, and will be only slightly tender. As the inflammation spreads to the peritoneum the tenderness will become greater, and the outline of the tumour will become less distinct—partly from the adhesions which it contracts, but mainly from the muscular rigidity which now appears. Pain will be continuous, but it varies in severity. In some cases there may be no elevation of temperature, but in others even at the commencement there are fever and malaise, and rigors may occur. In my experience the presence of fever has always been found to be associated with ulceration of the mucous membrane of the gall-bladder, and the pain

has been more severe than in those cases where the mucous membrane was intact. Where there are no constitutional symptoms the general condition of the patient may not deteriorate much, but if fever and great pain be present he will lose strength and weight.

Immediately the diagnosis of empyema of the gall-bladder has been made, cholecystotomy or cholecystectomy should be performed, and drainage of the abscess cavity must be continued till the discharge is sterile. If the condition of the patient is such as to permit of it, the cause of the obstruction in the duct should be removed; but, in some cases where operative interference has been delayed, especially in the aged, it may be well to content one's self with drainage of the cyst, and to leave the removal of the cause to a later date.

Phlegmonous cholecystitis is induced by a much more acute infection of the gall-bladder than that which gives rise to empyema, and consequently runs a much more rapid course, usually terminating fatally in a few days; though, if early operative treatment were in all cases adopted, there does not seem to be any reason why it should have a higher rate of mortality than has acute appendicitis. The gravity of the condition seems to depend on the fact that infective peritonitis is set up very rapidly before any localising adhesions have had time to form, the microbic contamination apparently arising without any direct gross communication between the interior of the gall-bladder and the peritoneum.

In these cases the walls of the gall-bladder become rapidly swollen, soft, and œdematous, the peritoneal coat loses its lustre, and there rapidly supervenes an acute peritonitis originating in the right upper part of the abdomen, leading to paresis of the intestinal coils located in that region, and giving rise to symptoms of intestinal obstruction. The gall-bladder will be more or less distended with muco-pus, and its surface will be purplish or even have a green tinge. If the patient survive, and nothing be done to relieve him, the gall-bladder will necrose in patches, but death may take place from septic peritonitis before this stage is reached. If death does not rapidly ensue, adhesions may form, limit the inflammatory disturbance, and lead to the formation of a localised abscess, which will follow a course similar to that of the abscess due to acute appendicitis.

Generally acute infective cholecystitis is associated with cholelithiasis, but it has followed on cholecystitis occurring during typhoid and other fevers, and in some cases without apparent cause.

Symptoms.—Either as a sequel to a prolonged history of "spasms," or apparently spontaneously, the patient is seized with sudden acute pain in the right hypochondrium radiating to the epigastrium, and through to the right subscapular region, which rapidly spreads till the whole abdomen becomes affected. Associated with this are the usual signs at first of local, later of general peritonitis. In the earlier stages there are tenderness and some distension below the right costal margin, with rigidity of the right rectus, but soon there comes general abdominal tenderness and tympanites, though even in the later stages the tenderness is most intense in the right hypochondrium. As occurs in other cases of acute peritonitis, there are a rapid thready pulse, quick thoracic breathing, and more or less collapse.

Before rupture has taken place the temperature will usually be found elevated, but when general infection has occurred the temperature curve may give little indication of the patient's condition. As a rule there will be no jaundice, or only a trace, as the disease progresses too rapidly to permit of its appearance, but in the more chronic cases it may be present.

Unless there is a previous history of gall-stone colic the diagnosis of the exact condition in phlegmonous cholecystitis is difficult, and usually it will only be possible to say there is acute peritonitis beginning in the right side of the abdomen. The site of the original pain and the direction in which it radiates, together with the greater tenderness in the right hypochondrium, and the primary appearance of distension in that region, may help one to come to a decision. Fortunately the conditions which it simulates—fulminating appendicitis and perforation of some hollow viscus—demand the same initial treatment, viz. exploratory laparotomy.

Treatment.—At the onset the pain is so severe that a hypodermic injection of morphine may be necessary. If the tenderness be not too great, local hot applications may be used, and all feeding by the mouth should be stopped.

Whenever there is a fair presumption that the case is one of phlegmonous cholecystitis, or, in the event of no accurate diagnosis being possible, it is found that the patient is gradually getting worse, and the evidence of peritonitis becomes more manifest, the abdomen should be opened. If gangrene has supervened, cholecystectomy should be performed; but if the disease has not progressed so far, it may be sufficient to aspirate the contents of the gall-bladder, then to open it and drain. The condition of the patient may be such that unless cholecystectomy can be easily accomplished it may not be wise to make any attempt to remove the disease at the first operation. Should there be much peritonitis it may be wise at the same time to drain the right kidney pouch, either by gauze introduced through the wound in the anterior abdominal wall, or by making a counter-opening in the loin. I have operated by cholecystectomy in a considerable number of cases with the happiest results, so that it would be only in very extreme cases that I should think it wise to divide the operation into two parts (9).

Croupous Inflammation of the Gall-Bladder and Bile Ducts.—Recently renewed attention has been directed to a form of cholecystitis and cholangitis associated with the formation of membrane or casts of the bile passages. The disease simulates in most respects ordinary cholelithiasis, in which a gall-stone is passing through either the cystic or common bile duct, the pain and other evidences of such a condition being quite marked. Occasionally in these cases, when the stools are being searched for gall-stones, there have been found either distinct membranous casts shaped like the gall-bladder, or flakes of membranous material. The condition is usually associated with membranous enteritis, but there are, in addition, the symptoms of gall-stone colic and consequent jaundice, caused by the passage of solid material through the inflamed ducts.

The disease may be associated with gall-stones, or may follow after a long history of cholelithiasis.

As a rule the diagnosis is impossible unless membrane be found in the motions, and the case will be considered to be due to cholelithiasis. This mistake is of less consequence since the treatment for the two conditions is the same. If, under the exhibition of saline aperients and careful regulation of the diet, the symptoms do not abate, cholecystotomy should be done, any gall-stones present removed, and drainage of the cyst established. At the same time any adhesions of the biliary apparatus to the surrounding viscera should be broken down. After recovery from operation the patient should be advised so to regulate his life as to diminish the risk of the occurrence of any gastro-intestinal disturbance or of cholangitis.

Cholangitis

Acute Catarrh of the larger Bile Ducts.—Catarrhal jaundice, so-called, is an affection which, for the most part, occurs in young persons, and usually results from dyspepsia or exposure to cold, but it may take origin from other causes such as pneumonia, the infectious fevers (especially typhoid), and cancer of the liver. That this condition is due to catarrh of the larger ducts in many cases is probable, but, as I pointed out in my Hunterian Lectures (8) on Diseases of the Pancreas, I think that so-called catarrhal jaundice is frequently due to a swelling of the head of the pancreas compressing the common bile duct which passes through it in 60 per cent of cases.

It is rare that uncomplicated cases succumb, but in some, where post-mortem examinations have been made, there has been found swelling of the mucous membrane, and plugs of mucus in the ducts have not infrequently been discovered. In addition, it is said, it can be seen that no bile has passed over the mucous membrane for some days, as all colour had disappeared from the affected part of the tube. Where death has taken place in cases of typhoid fever complicated with jaundice there has frequently been found unequivocal evidence of inflammation of the mucous membrane lining the bile passages. The catarrh is most marked in the common duct and gall-bladder, and gradually fades off in the hepatic ducts.

Judging from the appearances found post-mortem, as well as from the clinical histories, it may be inferred that the inflammation usually spreads from the duodenum. Even a very slight inflammation at the termination of the common duct in the posterior wall of the duodenum would suffice to block the passage of bile, since it is secreted under very low pressure.

Ordinarily catarrhal jaundice is preceded by some evidences of gastro-intestinal catarrh, either in the shape of a mild attack of dyspepsia with coated tongue and loss of appetite, or as an attack of nausea and vomiting with or without diarrhœa. In a day or two slight discoloration of the conjunctivæ appears, and the icterus increases for a week or ten days, thereafter gradually subsiding. Generally the patient feels out of sorts, but it is not usual for much constitutional disturbance to take place.

Where the catarrhal condition in the ducts complicates some other disease the course of events will depend on the nature of this illness.

In the majority of cases, where the patient is young and there is preceding gastro-intestinal disturbance, there will be little difficulty in arriving at a correct diagnosis in a case of simple catarrhal jaundice, but it should be remembered that slight jaundice is often a comparatively early symptom in cancer of the liver, it being then usually due to associated cholangitis.

The possibility of the disease being acute yellow atrophy of the liver should be kept in view, and indeed during the first week of this disease there may be no signs pointing to the gravity of the condition, and its course may exactly simulate an attack of simple jaundice. In the later stages of the disease, however, the urgent gastric symptoms and the associated delirium, with the rapid pulse and subnormal temperature, taken along with the progressive diminution in the hepatic dulness and the appearance of leucin or tyrosin, or both, in the urine can leave no doubt as to the diagnosis.

In jaundice due to cholelithiasis the presence of pain and tenderness, and the previous history of gall-stone colic or "spasms," will readily lead to a definite opinion being given, but if, as sometimes happens, there be no marked pain, the icterus will be found to pass off within a few days.

The jaundice of hypertrophic cirrhosis may closely simulate that from simple catarrh, but in this form of cirrhosis there is enlargement of the liver and usually other symptoms, such as ascites, pointing to the real origin of the icterus,

while the more advanced age of the patient and the previous history of alcoholism will help to prevent a mistake in diagnosis.

Generally little treatment is necessary in order to get rid of simple catarrhal jaundice. Since the infection originates in the duodenum, and is kept up by the condition there, it will be well to give some mild purgative such as calomel or a saline. The food should be of such a character as to be readily digested and not subject to early fermentative changes; no alcohol should be given.

Large rectal injections of hot water at a temperature of from 70° to 90° F. have been recommended, and are supposed to act by inducing active contractions of the gall-bladder, which expel the mucus blocking the common duct.

Chronic Catarrh of the larger Bile Ducts.—Though simple acute catarrh may assume a chronic phase, this is unusual, and chronic cholangitis, giving rise to symptoms, is usually due to some other cause. Of these the most common is probably the presence of gall-stones, but in many cases of cancer of the liver the jaundice is, in the earlier stages, due in the main to the catarrhal condition of the mucous membrane of the bile ducts, which is invariably present at all stages. Similarly, when jaundice is present in cases of hydatid of the liver, or of hepatic abscess, it is more frequently due to an inflammatory swelling of the ducts than to pressure on them by the tumour.

Where the process is merely a continuation of an acute catarrh the symptoms will be slight, consisting mainly of more or less icterus and some gastro-intestinal disturbance. The persistence of the jaundice may lead to a suspicion of serious organic disease, especially cancer, but as a rule there is not much loss of flesh or strength, and the jaundice does not progressively deepen as it does in malignant disease. Moreover, the symptoms will, more or less readily, yield to appropriate treatment in the simple cases, whereas in malignant disease there will be at the best only temporary amelioration.

Many cases of chronic catarrhal jaundice are undoubtedly due to interstitial pancreatitis, which when extreme may simulate cancer of the head of the pancreas, but which, unlike cancer, can be cured by a cholecystenterostomy, as I have proved on many occasions. A chemical examination of the urine and fæces enables a differential diagnosis to be made (7). Where the catarrh has been caused by gall-stones and persists after these have been passed, there is apt to be present in the ducts thick ropy mucus which has difficulty in passing along the narrowed ducts, and often gives rise to slight attacks of pain. Occasionally, as in cholecystitis, the mucus becomes much inspissated, and then when passing gives rise to pain resembling in almost all particulars that due to the passage of a gall-stone. So marked may be the resemblance, that in some cases it is not possible to differentiate between the two classes, but as a rule in cholelithiasis there is more marked tenderness, and for the most part some evidences of adhesions of neighbouring organs.

Chronic catarrh with or without jaundice should be treated, along the same lines as the acute affection, by salines (the best being the natural Carlsbad water), light diet, and regular exercise. With great care massage may be employed, and in the absence of gall-stones will probably be beneficial, but if there be any suspicion that the condition is directly due to cholelithiasis this treatment should be avoided. In the event of pain being present, topical remedies in the shape of hot fomentation, or the ingestion of a tumblerful of water as hot as it can be borne, will probably be sufficient; but it may be necessary occasionally to use sedatives. I have found half a drachm of spirit of ether in chloroform water, repeated every quarter of an hour if necessary, most effectual; but it may be necessary to give morphine hypodermically.

Unless cure or very marked relief follows this treatment within a month, the best treatment is to drain the gall-bladder and ducts by performing cholecystotomy or cholecystenterostomy, when if any gall-stones are present they may be removed at the same time.

Whether any calculi are found or not, the tube should be retained in the gall-bladder for some time; indeed, in the absence of stones it will probably be necessary to drain for a longer period, or to permanently short circuit the obstruction in the head of the pancreas by cholecystenterostomy. The best index to the time for withdrawal of the tube is sterility of the discharge; but roughly, it should be retained until the exudate is thin and contains little or no bile, as it is then clear that the normal secretion has been reached and the common duct is patent. After removal of the tube general treatment like that outlined above should be continued for some considerable time.

Infective Cholangitis.—In all forms of catarrh of the bile ducts there is probably some degree of microbic infection, and the peculiar symptoms which differentiate so-called "infective cholangitis" from ordinary catarrh are probably to be attributed to the occurrence of intermittent complete stoppage of the evacuation of the inflammatory products into the duodenum.

In the great majority of cases this will be found to be caused by gall-stones in the common duct either in the shape of a single stone "floating" in the duct and acting as a ball valve, or of multiple stones becoming impacted probably from some temporary swelling of the walls of the channels. Occasionally, however, malignant disease, either of the head of the pancreas and involving the orifice of the common duct or of the liver, is associated with infective cholangitis.

The symptom which specially characterises infective cholangitis is the occurrence of ague-like seizures, occurring at irregular intervals, and associated with remittent jaundice. The icterus in the interval between the attacks rarely completely disappears, though it may diminish so much as to be evident only on careful examination of the conjunctivæ in good light.

In a typical case there will be a more or less definite history of attacks of gall-stone colic, extending over, it may be, many years, and often without any marked degree of jaundice. Then comes a more severe attack, lasting longer, and followed by definite jaundice, probably denoting that the calculus has passed from the cystic into the common duct. After a short interval, during which the jaundice may have quite cleared, this is followed by another cholelithic seizure, accompanied by a rigor and all the symptoms of an attack of ague. Following on this there is jaundice which persists, though less in degree. This sequence of events recurs at irregular intervals, but in the majority of cases tends to return gradually more frequently, while the attacks become more severe. Even in the milder cases the strength of the patient progressively declines, and in the graver cases he may lose weight as quickly as if he were suffering from malignant disease of the stomach.

As a rule, this condition is not recovered from spontaneously, but occasionally the stone passes or ulcerates its way into the duodenum, and the patient gets well. It is, however, much more common for the course to be downward, the patient either succumbing to the repeated attacks of pain and poisoning, or to some complication of the disease, such as suppurative hepatitis, perforation of the ducts and peritonitis, endocarditis, or some pulmonary inflammation.

The diagnosis of this condition will not usually give much difficulty, at least in this country where ague is rare. The history of the cholelithic colic extending over some time, the pain specially localised in the neighbourhood of the gall-bladder and ducts, and the tenderness most marked midway between the xiphoid and the umbilicus, together with the progressive deterioration of health and the remittent character of the icterus, all help one to an accurate opinion. It is not common to find the gall-bladder distended, as it has usually become thickened from previous chronic inflammation, nor is it the rule for the liver to be enlarged, though if the suppurative condition extend into the finer ducts, or if the obstruction to the flow of bile is more or less complete, there will soon be some degree of hepatic enlargement. From the beginning, however, tenderness in the right hypochondrium and epigastrium is present.

Treatment should not be put off when once the diagnosis is at all established, as in the great majority of cases palliative treatment is of no avail, and valuable time will be lost since the patient will progressively lose strength.

If possible the cause should be removed by choledochotomy, but should this prove impossible, or the condition of the patient be such as to render it undesirable, the ducts should be drained and the cause removed by a further operation.

There can, however, be no doubt in the minds of those who have observed many cases that it is better to anticipate the complication, and as soon as medical measures, after a fair trial, have failed, to remove the gall-stones by surgical means.

Suppurative cholangitis, though in the great majority of cases caused by the presence of gall-stones, occurs also in association with cancer of the ducts, hydatid disease of the liver, and typhoid fever, and a number of cases have recently been reported in which the condition seems to have taken origin from the presence of ascarides in the common duct.

Associated with the causation in some obstruction in the ducts there comes dilatation to a greater or less degree of the lesser ducts in the liver, which also become very much inflamed. The whole liver enlarges rapidly, and may assume enormous proportions. Post-mortem the liver is large, and the ducts dilated and containing pus, while distributed through the liver there are usually found small abscesses of irregular shape.

Symptoms.—In the greater number of cases there will be a well-marked history of gall-stone colic, extending over a more or less lengthened period before the onset of acute symptoms, and it may be that there has been infective cholangitis; but where the disease is due to cancer, hydatid disease, typhoid, or any of the less common causes, the onset of the suppuration in the bile channels may be preceded by no local hepatic symptoms.

From whatever cause arising, the onset of suppuration is usually announced by a rise in temperature, with or without rigors or sweating, and this is followed by hectic temperatures, with rigors and profuse respiration.

Unless the attack has begun with a gall-stone colic, there may not be much pain at the beginning, but it is rare for some pain not to be present in the later stages. As with pain, so with tenderness; at first there may be little or none, but when the inflammation extends to the peritoneal covering of the liver the right hypochondrium becomes progressively more painful to the touch. Jaundice is present from the onset, and may become intense, but death as a rule takes place before this occurs. The liver enlarges uniformly, and may so increase in bulk that the lower margin descends below the umbilicus. The gall-bladder may enlarge, but as a rule it has become thickened and shrunken before the onset of the acute symptoms.

When the inflammatory disturbance implicates the peritoneum there come the usual symptoms of local peritonitis, viz. localised abdominal distension, with tenderness, and it may be vomiting. The pulse tends to increase in frequency and to lose in strength, the patient ultimately assuming the typhoid condition with dry tongue, shrunken features, rapid weak pulse, and great prostration.

In two cases of generalised suppuration in the liver (without any pulmonary complication) recently seen, the much greater proportional increase in the rate of respirations as compared with that of the pulse was a noteworthy feature, in each case the pulse-rate having gone up only 50 per cent, while the respirations had doubled in number.

Occasionally the disease pursues a subacute course, and then may end in a localised hepatic abscess, but this is extremely rare.

Death usually takes place from exhaustion, but may be accelerated by the occurrence of various complications, of which the commonest seem to be pyæmic abscesses, pleurisy, pneumonia, and infective endocarditis.

Pylephlebitis pursues a similar course, and it may not be possible to distinguish it from an attack of suppurative cholangitis unless there be a definite history pointing to the existence of some focus of suppuration in the parts drained by the portal vein.

In the earlier stages where the diagnosis is not clear the patient should be given 5 grs. of calomel, followed by a seidlitz powder, and local warm applications should be used, while if the pain is extreme it will be necessary to give sedatives; but whenever the case is made out clearly to be one of suppuration in connection with the bile passages, operation should be resorted to, since it is only in the early stages that one can reasonably hope for complete recovery.

As a rule it will be advisable only to attempt to give exit to the inflammatory products, and to establish drainage by performing cholecystotomy, leaving the cause to be removed at a later time. But if the patient be in fair condition, and the exciting cause of the malady can readily be reached, that should be got away at the same time.

If performed early, the relief to tension and the evacuation of the septic contents of the gall-bladder and bile ducts should give a very fair probability of complete cure, but even in the later stages, unless indeed the patient is moribund, it is well to seek relief by drainage, as some cases that appeared almost hopeless have recovered, and in any case the symptoms will be ameliorated.

Drainage should be kept up till the discharge becomes sterile.

Adhesions

Among the most distressing sequelæ of gall-stones, when these have been present for a long time, are the results which follow on the contraction of inflammatory adhesions to neighbouring viscera. The organ which is most frequently involved is the stomach, as the proximity of the duodenum and pylorus to the cystic duct renders it specially liable to suffer.

Usually the pylorus itself is tucked up closely to the cystic duct by adhesions, often so short as to render it a matter of extreme difficulty to separate the two without perforation of either viscus; but, not infrequently, there is merely some kinking of the pyloric extremity of the stomach or of the duodenum by adhesions to the gall-bladder or cystic duct.

Less commonly the whole, or part, of the lesser curvature of the stomach becomes adherent to the under surface of the liver.

Whichever of these conditions is present, there follows the whole series of symptoms which arise when, from any cause, the stomach is unable to empty its contents into the duodenum within the normal time. When the stomach wall over a considerable area is adherent to the liver, but there is little or no implication of the pylorus, the symptoms will only amount to some degree of dyspepsia and a feeling of discomfort in the epigastrium, coming on shortly after food and lasting for several hours, since there is no actual obstruction to the outflow from the stomach, but only interference with the peristaltic wave. When, however, the pylorus becomes constricted by surrounding adhesions, or the exit from the stomach is obstructed by kinking, there follows dilatation of the stomach, giving rise to the well-known symptoms of that disorder which will prove most intractable to ordinary medical remedies, and even to lavage. If left untreated by operation this condition exhibits no tendency to improve; but, as a rule, despite the most careful and continued treatment, the condition of the patient becomes progressively worse, the stomach dilating so as to reach almost to the pubes, vomiting, even after liquid food, taking place after each meal, and the patient becoming worn out from the combined effect of defective nutrition and continued pain. The frequency of this condition is not yet quite realised by most practitioners, though those who see much of gall-stone work must have noted the large proportion of cases in which it occurs to such an extent as to be readily diagnosed, before operation, by the usual physical signs of dilatation of the stomach.

It should be noted that, when the pylorus is adherent to the cystic duct, and there are biliary calculi in the duct or gall-bladder, the pain of a cholelithic seizure may radiate to the *left* subscapular region instead of to the right.

Less commonly, but still not infrequently, the colon is obstructed to a greater or less degree by the presence of short adhesions to

the gall-bladder, or by larger bands resulting from localised peritonitis so pressing on the bowel as to interfere with the circulation of its contents. As might be expected, the middle part of the transverse colon is the usual seat of obstruction, but I have seen the hepatic flexure blocked, and in one case recently treated for acute obstruction there were two marked bands, one situated almost at the hepatic flexure, the other within a couple of inches of the cæcum.

Where the colon is involved the symptoms are as a rule less severe than when the stomach is affected, but, when bands have formed, acute intestinal obstruction may rapidly develop. As a rule, however, there are only evidences of chronic intestinal obstruction, showing itself by attacks of paroxysmal colicky pains in the abdomen, occurring at irregular intervals and associated with constipation, or constipation alternating with diarrhœa. Vomiting is not commonly present, but, when the kinking is acute or the bowel is constricted by bands, this may be a prominent feature. In many of these cases during the attacks of pain careful examination will reveal the cæcum contracting in its efforts to force the contents of the colon through the stricture or past the kink; and at times large fæcal concretions can be felt on the proximal side of the obstruction.

In all these respects the case may closely simulate cancer of the large intestine but in the latter there is more rapid deterioration of the general health, and since the stricture in malignant disease is most often in the sigmoid flexure or rectum, the motions tend to be flattened, and there is straining and the presence of mucus and blood in the stools.

When gall-stones give rise to local peritonitis so marked as to lead to obstructive symptoms, there are always long-continued and repeated attacks of colic, so that the past history is of great importance in arriving at a correct conclusion in any individual case, though it should be remembered that cholelithiasis and cancer may of course be present at the same time.

In most cases of adhesions there is more localised tenderness than when the obstruction is due to new growth, but the attacks of paroxysmal pain may be as severe in one case as in the other.

Where adhesions are diagnosed, or where, after a history of repeated attacks of gall-stone colic, symptoms arise which might be referable to adhesions, and which do not subside under medical treatment sufficiently long continued, operation should be undertaken, and the adherent viscera separated. As a rule there will be gall-stones present, but, in any case, it will probably be advisable to perform cholecystotomy and drain the gall-bladder to get rid of the catarrhal condition of the mucous membrane, which is almost invariably present. In order to prevent the recontraction of the adhesions the omentum should be tucked up between the gall-bladder and the contiguous viscera.

After recovery from operation the patient should be advised to continue medical remedies directed to prevent catarrh of the duodenum and bile duct, as has been advised after cholecystotomy for gall-stones.

Ulceration and its Sequelæ

Ulceration of the gall-bladder and ducts is probably fairly frequent, but if uncomplicated does not usually give rise to symptoms of much importance. Although cholelithiasis is the most frequent, typhoid fever and cancer are quite common causes, and tubercular disease and cholera also produce ulceration.

As has already been pointed out, pyrexia when present in empyema of the gall-bladder is probably always associated with ulceration, and due to absorption of septic products through the ulcerated surface.

The importance of ulceration, however, depends more on its sequelæ, the most important of which are adhesions (already considered), perforation, fistula, peritonitis, hæmorrhage, and stricture.

As the ulceration extends through the wall of the cyst or its ducts, there is set up local peritonitis, which usually induces adhesions to the neighbouring viscera, and thus shuts off the general peritoneal cavity, but occasionally no adhesions are formed, and perforation takes place. On account of the fact that the bile in cases of cholelithiasis is invariably infected, this event is always very serious, since general septic peritonitis is almost certain to ensue, and unless quickly treated will end fatally. Rarely the perforation is followed by a localised peritonitis, which shuts off the general peritoneal cavity, and allows of the formation of an abscess which runs a course similar to that of an abscess due to appendicitis.

The symptoms of perforation of the bile passages are those of perforative peritonitis from any cause, with, as a rule, a history pointing to the presence of gall-stones. A sudden pain beneath the right costal margin followed by collapse and succeeded by vomiting, general distension, and a rapid pulse, form the chief features of the disease. If the extravasation is extensive there will be signs of free fluid in the peritoneal cavity, and jaundice, if not present before, usually appears within twenty-four or forty-eight hours from absorption of biliary pigment by the peritoneum. Death takes place usually within a few days, but cases have been reported where life was prolonged into the second or third week.

As a rule the condition of the patient does not warrant more than that the abdomen should be opened, the extravasated material removed by sponges, and satisfactory drainage established; but, if his condition be sufficiently good,

the perforation should be found and stitched, gall-stones if present removed, and the gall-bladder drained separately from the general cavity of the peritoneum.

Fistula.—Fistula is a not infrequent complication of ulceration of the gall-bladder and bile ducts. Much more frequently the communication is between the surface of the body or the cavity of one of the hollow viscera and the gall-bladder, but communication with the larger ducts is not uncommon. In the direct variety, where the channel is formed directly through local adhesions set up by the advancing ulceration, the communication is most frequently with one of the hollow viscera; whereas, in the indirect variety, caused by the formation of an abscess outside the gall-bladder and a channel forming from this, the fistula more usually opens on the surface of the body.

As might be expected from the anatomy of the parts the structures most commonly affected are the duodenum and colon; but fistulæ have been described between the biliary passages and the stomach, the jejunum, the ileum, and the pelvis of the right kidney, while a number of cases have been described in which gall-stones have perforated into the thorax.

Biliary cutaneous fistula, like the other forms of fistula in connection with the bile passages, is most commonly a sequel of gall-stones, but may result from any of the causes which give rise to perforation of the bile channels, especially when the perforation leads first to a localised abscess. When arising from suppuration in connection with the gall-bladder or bile ducts the fistula usually opens near the umbilicus, the pus following the course of the obliterated umbilical vein, but the discharge may occur at any part of the abdominal wall. Occasionally a fistula persists after operation where the obstruction in the ducts has not been overcome, or the gall-bladder has been stitched to the skin instead of to the peritoneum and aponeurosis. Their importance varies according as only mucus or bile and mucus are discharged, since in the former case only about one ounce of fluid is discharged daily, and this does not give rise to much inconvenience.

Mucous fistula occurs when the cystic duct is obstructed by the presence of a foreign body, or is occluded by stricture resulting from old ulceration.

So long as there is free exit to the secretion there will only be the inconvenience arising from the necessity of constantly wearing some dressing to absorb the discharge; but if the orifice of the sinus be allowed to close, the accumulation produces pain, and it is necessary under these circumstances for the patient either to wear a small drainage-tube and an absorbent dressing or to submit to operation.

Should operation be decided on, the course pursued will depend on the cause of the condition. If there be an impacted calculus in the cystic duct, this should be removed; but if the continuation of the discharge depends on stricture of the duct, the gall-bladder should either be excised or connected with the duodenum by means of a Murphy's button. As a rule, in the latter case, cholecystectomy will be safer, since the gall-bladder, under these circumstances, is frequently small, and cholecystenterostomy in a case where there is a shrunken gall-bladder is always difficult of performance.

Biliary fistula is a much more serious matter, because, in addition to the disability caused by thirty ounces or more of bile being discharged on to the skin daily, there is apt to be interference with the general nutrition.

This form of fistula arises from some obstruction to the flow of bile in the common duct, most commonly from an impacted stone, but occasionally from malignant disease of the head of the pancreas or of the common duct.

Where possible the obstruction should be removed; but when it arises from malignant disease of the pancreas this is impossible, though when due to gall-stone in the common duct, it is as a rule advisable to perform choledochotomy. In the event of it being decided not to remove the obstruction, the best course open is to connect the gall-bladder with the duodenum, by performing cholecystenterostomy.

Before opening the peritoneal cavity in any case of biliary cutaneous fistula it is well to purify the sinus as well as possible by curetting the granulating track.

In a case definitely known to be due to a gall-stone in the ducts, before resorting to operative interference it may be well first to try for some time the effect of injecting olive oil, or a ·5 per cent solution of sapo animalis, by means of a flexible catheter introduced into the sinus, on to the obstruction two or three times daily, in the hope that by this means the concretion will be dissolved.

Biliary-Intestinal Fistula.—Fistula between the bile ducts and some part of the gastro-intestinal tract is a fairly common complication of ulceration due to gall-stones, and frequently arises without any overt manifestations, the ulceration proceeding slowly through adhesions into the lumen of the stomach or gut, the gall-stones being discharged and the fistulous track healing spontaneously. In several cases which I have seen, and in others reported, the first indication of anything having happened has been the onset of acute intestinal obstruction from the gall-stone becoming impacted low down in the bowel. But the process is not always accomplished without symptoms, such as pain over the liver, more or less jaundice, a fever of irregular character, with, it may be, some hæmorrhage into the stomach or bowel, while there is always the possibility of perforative peritonitis. Apart from the complications spoken of and the subse-

quent adhesions, the formation of such a fistula does not usually cause much inconvenience or give rise to any need for operative interference, but when the communication is with the stomach the passage of bile into that organ may lead to the necessity for active treatment. Rare forms —biliary-urinary, biliary-vaginal, biliary-thoracic, biliary-pulmonary, biliary-pericardial, biliary-mediastinal, biliary-pleural, biliary-retroperitoneal, and biliary-portal—have been described, but they are of extremely infrequent occurrence.

Stricture of the bile ducts, apart from that due to cancer, is, judging from reported cases, a much rarer condition than might be supposed. Though there is no reason why it should not be a sequel of ulceration arising from other causes than cholelithiasis, I am not aware of any case having been reported where it was not preceded by the presence of biliary calculi. It should be noted, however, that it may not declare itself until some time after the exciting cause has been removed, either by operation or through ulceration leading to the formation of a fistula through which the stones escape.

If in the cystic duct the only symptom present may be a gradually enlarging tumour in the gall-bladder region, with or without pain or uneasiness; if in the common duct, jaundice supervenes, at first being only slight, but ultimately becoming intense, the liver progressively enlarges, and if it be not shrunken from the continued irritation of gall-stones, the gall-bladder distends.

So far as I know, only one case of stricture of the hepatic duct has been described, and in it the symptoms were like those of stricture of the common duct, but with no distension of the gall-bladder. The case ultimately proved fatal from peritonitis due to ulceration of a stone into the peritoneal cavity.

Cases of stricture of the ducts can scarcely be diagnosed, but will mostly be suspected to be cases of impacted calculus, the condition only being recognised when the abdomen is opened.

In stricture of the cystic duct probably the best treatment is to remove the gall-bladder, but cholecystenterostomy may be performed.

When the obstruction is in the common duct, cholecystenterostomy or choledochenterostomy should, as a rule, be done, but if the patient be too ill to bear a prolonged operation, cholecystotomy will relieve the symptoms. It may occasionally be possible to excise a stricture and join the cut ends of the duct, or to perform a plastic operation so as to widen the lumen of the bile passage.

Unless there is great dilatation of the hepatic duct no operation is likely to be of much avail in stricture of that channel, but if it is sufficiently distended it might be feasible to connect it either with the gall-bladder or with the intestine by means of a Murphy's button.

Stricture of the gall-bladder, giving rise to an hour-glass shaped organ, occasionally occurs from ulceration of the gall-bladder, and may even proceed so far that the upper cavity is quite shut off from the lower. In such a case the upper sac should be amputated and the lower drained, as in ordinary cholecystotomy, after any concretions which may be present have been removed.

Peritonitis of an acute kind may occur along with the ulceration apart from any gross communication between the interior of the bile passages and the peritoneum, the removal of the epithelial lining apparently permitting microbes to pass through the wall of the sac, but this is not common. Another uncommon complication of ulceration is *hæmorrhage*. Usually as the ulceration proceeds thrombosis takes place in the vessels, but occasionally severe hæmorrhage results, and several cases have been recorded in which the fatal issue seems to have been determined by loss of blood arising in this way.

Tumours of the Gall-Bladder

Under this general head it is convenient to consider two very different conditions, viz. enlargement from distension and new growths affecting the gall-bladder.

The gall-bladder may, from a variety of causes, be so distended, apart from any new growth, as to be felt on palpation of the abdomen. The commonest cause is undoubtedly obstruction to the cystic duct by the impaction of a gall-stone or from hydatid disease, but it may follow on the blocking of the common duct by a stone or from pressure on the ducts exercised from without. The proportion of cases in which cholelithiasis is associated with distended gall-bladder is, however, comparatively small, as mostly, where gall-stones have been present for some time, they cause so much inflammatory thickening of the wall of the cyst and destruction of its mucus-secreting glands that it is incapable of much distension even should the ducts become blocked,—indeed it is much more common to find it shrunken and smaller than normal.

Where malignant disease is so situated as to occlude the larger bile passages, it is very much more common to find the gall-bladder so distended as to be palpable, because there has, as a rule, at least when the cause of the obstruction is situated in the common duct as it most frequently is, been no preceding interference with the glands studding the mucous membrane of the gall-bladder, and these continue to pour out mucus while the normal walls of the cyst readily yield to the increased internal tension.

A distended gall-bladder rarely reaches much below the umbilicus, but some have been found to fill the abdomen to such an extent as to have been operated on under the assumption that

they were ovarian tumours, and in one case the fundus of the gall-bladder was discovered in a femoral hernial sac. Except in those cases where the obstruction is in the cystic duct, the contents are probably in all cases at first bile with a small percentage of mucus, but later the bile becomes absorbed, and only mucus is present unless acute inflammation supervenes, when muco-pus will be formed.

A distended gall-bladder is to be made out as a pear-shaped body passing downward and forward from the ninth costal cartilage towards the middle line just below the umbilicus and moving with respiration. Where there has been little local peritonitis it will be felt quite distinctly as a smooth, rounded, and tense tumour, the lower extremity freely movable from side to side, but becoming more fixed and less defined as the liver is reached. In such a case in a thin patient the mass can usually also be seen to move up and down with expiration and inspiration, but if there has been local inflammation this will not be so, and the tumour will be found much less defined and probably more fixed. If the gall-bladder is inflamed there will also be a greater or less degree of tenderness, whereas in simple enlargement the mass will be painless on manipulation.

Partly on account of its tenseness, but mainly by reason of its mobility, fluctuation cannot usually be obtained, while absence of dulness on percussion is not uncommon from the presence of coils of intestine overlying the tumour.

In some cases I have found it easier to be sure of the presence of a distended gall-bladder by making the patient assume the genu-pectoral position, when it can be readily felt lying on the flat hand placed on the right side of the abdomen.

The detection of variations in size from time to time is of great importance, as it practically invariably denotes that the cause of the obstruction is simple, whereas in malignant disease once distension has occurred it persists.

Jaundice may or may not be present along with the palpable gall-bladder, according to the site of the obstruction, but when present it is a grave sign by reason of the consequent interference with nutrition, and from the fact that the two conditions are more usually associated with malignant disease than with cholelithiasis.

Pain is not commonly marked after the onset; but the beginning of the condition, when due to biliary calculi, is usually ushered in by an attack of gall-stone colic, while when arising from malignant disease there is for the most part no severe pain.

The commonest error in diagnosis is to assume that a distended gall-bladder is a floating kidney, or a renal or supra-renal tumour, and the mistake is more readily made than might be expected. With care, however, this can usually be easily enough avoided. Where the obstruction is in the common duct there should be no difficulty, since the presence of jaundice will readily localise the site of the disease. When the obstruction is in the cystic duct the case is not so easy, but the fact that the tumour is continuous above with the liver, that its lower extremity only is movable, and its range of movement small, that it is pear-shaped and not kidney-shaped, that though movable from side to side it cannot be displaced downwards at all other than by the patient taking a deep inspiration, and that when left alone it does not tend to fall towards the lumbar region, should render the diagnosis clear. Should there have been attacks of severe pain at any time the different characters of renal and gall-bladder pain, both as regards site of origin and distribution, will help to define the case.

In case of difficulty, which will occur mostly in very stout persons, or in those who are unable to relax their abdominal muscles, examination with the patient under an anæsthetic may be necessary. Only rarely will it be necessary to apply Ziemssen's test, which consists in distending the colon with gas, and so displacing a kidney tumour into the loin, or a distended gall-bladder toward the liver at the junction of the right hypochondrium with the epigastrium.

Where the kidney or supra-renal tumour has become adherent to the colon this test if relied on may give a false impression, as the mass may be raised very much into the position usually assumed by a distended gall-bladder.

Primary malignant disease of the liver in its early stages may closely simulate distension of the gall-bladder in its physical characters, but the greater irregularity of the surface, the history of the illness, the rapid loss of weight and strength before the formation of a perceptible tumour, and the subsequent onset of jaundice, will assist an accurate diagnosis. Hydatid disease is more likely to give rise to difficulty, but it is not so well defined, is not pear-shaped, and is as a rule painless throughout its history, while generally fluctuation or the characteristic thrill can be detected.

Tumours of the intestine and of the pylorus should be kept in mind when examining a case of suspected gall-bladder tumour, but the associated symptoms will usually enable one to make the diagnosis. In this connection it should be remembered, however, that dilatation of the stomach is very frequently a result of the pylorus becoming adherent to the cystic duct in cholelithiasis.

The possibility of the tumour being a Riedel's lobe should be remembered, but when this abnormal projection is present it is usually farther to the right, its shape differs from that of a gall-bladder, being broadest at its upper extremity and tapering off downwards, while it

is harder and firmer. At times, too, the gall-bladder can be felt to its inner side.

The treatment of distended gall-bladder will depend on the cause, but as a rule, when due to non-malignant disease it will be necessary to perform cholecystotomy, and at the same time remove the obstruction whether that be situated in the cystic or common duct. When due to stricture of the cystic duct the gall-bladder should either be removed, or connected to the duodenum by the operation of cholecystenterostomy.

New growth affecting the bile passages is almost always of the nature of columnar-celled epithelioma, but simple tumours have been found from time to time, and some believe that adenomata precede all primary carcinomas affecting the gall-bladder or ducts.

Primary cancer of the gall-bladder is not very common; but it does supervene on chronic cholelithiasis more often than is usually believed, as when the disease occurs it is quite commonly assumed to be primary malignant disease of the liver. More frequently the gall-bladder becomes affected with cancer by extension from neighbouring organs, and then the tumour differs in its histological characters according to the site of origin. When the gall-bladder alone is affected as a rule the wall is uniformly infiltrated before the disease is sufficiently marked to give rise to symptoms. Dissemination, otherwise than by local extension, is rare, but the glands in the lesser omentum usually are involved in the later stages. Mostly the liver is the organ first invaded, but where cholelithic symptoms have been marked it is not unusual for the pylorus, duodenum, or colon to be involved early on account of their having become previously adherent to the gall-bladder.

The diagnosis of cancer of the gall-bladder is always difficult in the early stages, and often impossible before exploration of the abdomen.

Almost invariably there is a history of attacks of gall-stone colic extending over a lengthened period, then comes progressive deterioration of health without much local manifestation other than a sense of discomfort in the gall-bladder region scarcely amounting to pain. As the disease progresses the pain becomes more marked and more diffuse, often extending toward the right scapular region.

In the early stages no tumour can be made out on palpation, but with the growth of the local disease a hard rounded mass comes to be readily felt below the costal margin, at first freely movable during respiration, but later becoming more fixed and nodular. Unless gall-stones are present there is throughout an absence of anything like marked tenderness.

Early in the disease there is no jaundice, but later jaundice is frequent, and may be due to extension of the disease along the cystic duct, to catarrh of the ducts, to invasion of the liver, or to pressure on the common duct by the enlarged lymphatic glands.

Associated with these symptoms may be others arising from the extension of the growth to the surrounding organs.

As the disease progresses, the general condition of the patient rapidly deteriorates until he succumbs to the malady.

Malignant disease may be closely simulated by inflammatory adhesions in the neighbourhood of the gall-bladder, but in the latter case there is not the same deterioration of health unless suppuration supervenes, when the elevation of temperature and marked local tenderness will afford a clue to the state of affairs.

Where the disease is limited to the gall-bladder, treatment by cholecystectomy affords a very fair hope of cure; and even if the liver be involved, in certain cases it will be found possible to remove the disease with a possibility of cure, and the certainty of amelioration of symptoms. In the majority of cases, at the present time, however, when the patient comes to the surgeon nothing but the alleviation of symptoms by the exhibition of sedatives can be hoped for.

Tumours of the Bile Ducts

When the gall-bladder is much shrunken from old-standing inflammation it occasionally happens that the common duct distends so markedly, as a result of obstruction low down, that it can be made out as a distinct tumour.

As a rule these cases have been diagnosed either as distended gall-bladders or as hydatid disease of the liver, and the true state of affairs has only been made out when the abdomen was opened. If such a case be due to an impacted gall-stone this should be removed, and the opening in the duct through which it is extracted stitched up, since the cases which have been treated by drainage of the duct have nearly always up to the present done badly. Where it is not possible to remove the cause, choledochenterostomy should be performed rather than choledochostomy, since a permanent biliary fistula will follow the latter procedure, while the chance of recovery from operation, judging from published results, is distinctly less than when the bile stream is short circuited.

Simple *new growths* of the bile ducts occur, but are very rare; malignant disease, though not at all a frequent condition, is much more common.

Any part of the larger ducts may be involved, but the tumour is most frequently situated in the lower part of the common duct.

Practically in all cases cancer of the ducts is preceded by cholelithiasis, and this renders the diagnosis difficult, as the symptoms of both conditions are usually present.

The growth is usually annular, and histologically is a columnar-celled carcinoma. The

special symptoms depend on the fact that the outflow of bile is readily obstructed; and thus when situated in the common duct malignant disease gives rise early to jaundice which progressively deepens, the liver gradually increases in size, and the gall-bladder distends, unless previously shrunken as the result of the presence of gall-stones.

Infective or even suppurative cholangitis not infrequently supervenes, and gives rise to the symptoms already described. Pain may be present, even though the gall-stones have passed; but, where none are present, there is as a rule little or no tenderness, but only a feeling of discomfort on deep pressure in the epigastrium.

Should the cystic duct be the site of origin of the disease the gall-bladder will distend, but jaundice will not be present to any degree until the growth has by extension invaded the common or hepatic duct, though even in the early stages slight icterus may be noted from a catarrhal condition extending from the stricture. Almost invariably gall-stones are present in the gall-bladder or cystic duct, and these may give rise to pain of a paroxysmal character and to tenderness.

The hepatic duct is comparatively seldom the first to suffer, but when it does the symptoms will be those due to obstruction of the common duct, but for the fact that of course there will be no distension of the gall-bladder.

It should be remembered that obstructive jaundice frequently arises from the extension of malignant disease from neighbouring organs, especially from the head of the pancreas, and while it may be impossible to differentiate the two conditions, as a rule there will be some symptoms pointing to the origin of the disease outside the ducts. The diagnosis may be materially assisted by a careful examination of the urine and fæces (7).

Only in tumour of the cystic duct at or shortly after the onset of the mischief in the common duct can radical treatment by removal of the disease be attempted. Where the hepatic duct is involved, surgical interference is practically of no avail; but when the obstruction is in the common duct, two courses of surgical treatment may be followed with relief to symptoms. Either cholecystotomy may be performed and a permanent biliary fistula established, or cholecystenterostomy may be done. Should the patient be in fair condition, and the gall-bladder distended or normal in size, the latter alternative should be adopted; but if he is weak, this operation becomes very dangerous, since it is necessarily more prolonged than a simple cholecystotomy, and in the event of the gall-bladder being less than its normal size should not be attempted.

Too often, however, before the patient comes under the surgeon's care he is so reduced that no operative measures are justifiable, and only remedies directed to the amelioration of his sufferings can be advised.

LITERATURE.—1. Courvoisier. *Beiträge zur Pathologie und Chirurgie der Gallenwege.* Leipzig, 1890.—2. E. M. Brockbank. *On Gall-stones.* Manchester, 1896.—3. H. D. Rolleston. "Carcinoma of large Bile Ducts," *Medical Chronicle*, Jan. 1896, and *Clinical Journal*, April 7, 1897.—4. Mignot. *Archives générales de médecine*, Aug. 1898.—5. Christian Fenger. "Stones in the Common Duct and their Surgical Treatment," *American Journal of Medical Science*, Feb. and Mar. 1896.—6. Complete references will be found in *Diseases of the Gall-bladder and Bile Ducts*, by A. W. Mayo Robson, 3rd edition, 1904.—7. A. W. Mayo Robson. *Edinburgh Med. Journ.*, Dec. 1905.—8. *Idem.* "Hunterian Lectures on Diseases of the Pancreas," *Lancet*, March 19 and 26 and April 2, 1904.—9. *Idem.* "Indications for Removal of the Gall-bladder," *Brit. Med. Journ.*, 1906.—10. *Idem.* "A Series of 123 Cases of Choledochotomy for Calculi in the Common Duct," *Medico-Chirurgical Trans.*, vol. lxxxviii.—11. *Idem.* "Modifications and Improvements in Operations on the Biliary Passages," *Brit. Med. Journ.*, Jan. 24, 1903.

Galloping Consumption. *See* Lung, Tuberculosis of (*Clinical Varieties and Progress, Acute*).

Gallop Rhythm. *See* Heart, Myocardium and Endocardium (*Symptomatology, Muscle Failure, Bruit de Galop*).

Galls.—Excrescences on *Quercus infectoria* caused by the eggs of *Cynips gallæ tinctoriæ.* They occur as small, hard, round, tuberculated masses of a dark green colour. They contain 60-70 per cent *tannic acid*, and 2-5 per cent of *gallic acid. Preparations*—1. Unquentum Gallæ; 2. Unquentum Gallæ cum Opio. The astringent action of galls is entirely due to the presence of tannic acid (*q.v.*). The ointment of galls and opium is an excellent remedy for inflamed or painful hæmorrhoids.

Gall-stones. *See* Gall-Bladder and Bile Ducts.

Galton's Law.—The law of ancestral inheritance: "the two parents between them contribute on *the average* one-half of each inherited faculty, each of them contributing one-quarter of it; the four grandparents contribute between them one-quarter, or each of them one-sixteenth," and so on in an infinite series, in which each term is equal to the sum of all those that follow. *See* Heredity.

Galton's Method of Identification.—The finger-print system. *See* Anthropometry; Medicine, Forensic (*Identity*).

Galton's Ventilating Fireplace.—A method by which an open fire is utilised for heating the fresh air which enters the room; the entering air passes over the back portion of the fireplace (where it is heated) and up a special shaft (not communi-

cating with the smoke flue) to open into the room near the ceiling. *See* VENTILATION AND WARMING (*Extraction System*).

Galton's Whistle. *See* AUDITORY NERVE AND LABYRINTH (*General Diagnosis, Localisation of Seat of Lesion*); EAR, MIDDLE, CHRONIC NON-SUPPURATIVE DISEASE (*Diagnosis of Middle Ear from Internal Ear Disease*).

Galvanic Cautery. *See also* CAUTERY. —The galvanic cautery is an instrument for burning tissues, the heat being produced by a current of electricity.

PRINCIPLE OF CONSTRUCTION.—A piece of metal of relatively high resistance—platinum being most commonly used—is placed in a circuit of ordinary copper wire. The copper wires are carefully insulated and bound together, being prolonged into the handle of the instrument. When the current is allowed to pass, the platinum, offering resistance, becomes heated, and thus forms the cautery. The handle is provided with a key by which the current may be easily closed and opened.

SOURCE OF CURRENT.—The current may be obtained from (1) a battery; (2) an accumulator; or (3) a lighting main.

1. *Battery.*—If a battery be employed for this purpose it must be different from that used for galvanisation or for electrolysis. In the latter cases high electromotive force, that is, high voltage, is required to overcome the high resistance of the human body, whereas a strong current cannot be borne. On the other hand, a strong current is required to render incandescent a piece of platinum of the thickness necessary for a cautery, and to produce such a current and maintain it constant, even for a short time, large cells are necessary; but as the resistance of platinum wire is very small relatively to that of the human body, a small electromotive force is required, therefore few cells.

A battery to be used for the sole purpose of providing current for a galvanic cautery should consist of from two to six bichromate cells of large size, the square shape being preferable to the bottle shape because it admits of the plates being farther apart, which secures greater constancy of the current. These cells should be connected "parallel," *i.e.* zinc to zinc and carbon to carbon, for by this arrangement the internal resistance is diminished and a stronger current is yielded. For the ordinary small cautery the battery must be able to produce a current of at least eight ampères.

2. *Accumulator.*—Generally the most convenient and the most reliable and constant source of current is the accumulator. It does not easily get out of order, and may be simply adapted for use either with the cautery or for a small electric lamp. Two or three secondary cells, each giving two volts, enclosed in a strong oak case, will be found most convenient for general purposes.

3. *Lighting Main.*—Both continuous and alternating sources of electrical supply may be employed for cautery work.

With a *continuous current* a suitable resistance, consisting of spirals of thick wire, must be provided. As the pressure in these mains varies from 100 to 250 volts a large amount of energy is lost, seeing that only two to four volts are required in the cautery. With a properly constructed resistance all dangers from accident are avoided, and, as the current is usually required for a very short time, the cost is not serious.

An *alternating current* affords a very simple and economical means of heating a cautery. All that is necessary is to insert in the circuit the primary of a small transformer, the secondary of which is wound so as to produce a large current at a small pressure. The strength of the current in the cautery is regulated by the position of the two coils relatively to each other. An excellent transformer, suitable for this purpose, is that devised by Woakes.

FORMS OF INSTRUMENT IN USE.—In work on the nose and throat and in dental work are found the commonest applications of the galvanic cautery. The handle devised by Dr. Schech is the one most commonly used. To this any of the platinum points, or "burners," of which there are many forms—probe-pointed, flat, spear-shaped, etc.—differing in size and shape, may be fitted; or a wire, arranged as a snare or écraseur. For the latter purpose the ordinary steel wire, of various thicknesses, which is used for pianos, is quite suitable.

When a larger heated surface is required a piece of porcelain may be employed, introduced within a loop or spiral of platinum wire, which with the wire becomes heated when the circuit is closed.

DISEASE IN WHICH THE GALVANIC CAUTERY IS EMPLOYED.—*Throat.*—In hypertrophic pharyngitis, for the destruction of hypertrophied tissue. It may sometimes be applied to hypertrophied tonsils. In the larynx it has been employed for the destruction of small neoplasms; or, as a snare, for the removal of larger pediculated tumours.

Nose.—Again, for the destruction of hypertrophied tissue causing obstruction of the nares. Here the flat burner is employed; but the snare may also be used to remove large masses, especially when they are attached by broad bases, because the hot wire makes for itself a furrow, and obtains thus a hold which the cold wire may fail to do. It is also useful for cauterising pedicles of polypi removed by the cold snare or by forceps. And in cases of epistaxis due to an unhealthy condition of the mucosa its application, at a dull red heat, is very valuable.

Ear.—Here the applications are fewer, but the galvanic cautery may be used for the removal of redundant tissue in the meatus, or for perforating the drum membrane, in rare cases. It has also been used for destroying foreign bodies impacted in the meatus, as peas, etc.

Gynecology.—Most frequently used as a snare for amputation of a hypertrophied cervix uteri. But it is also sometimes employed as a cautery for destroying urethral caruncles.

Genito-urinary Surgery.—For the radical cure of the symptoms which arise in cases of enlarged prostate a special apparatus has been devised by Professor Bottini. It requires a very strong electric current.

General Methods, etc.—Local anæsthesia should always be induced before the cautery is used, by means of pledgets of cotton wool, saturated in cocaine (20 per cent), and placed in contact with the affected part for a few minutes before the application is made.

The operator must be careful to test the apparatus before using it. All the screws and connections must be clean and firm, and the wires used for connections strong and well insulated. The current must be gradually strengthened till the burner is brought to a white heat. (In contact with the tissues this will just procure the necessary degree of redness.) If this be not done the point may fuse whilst in use, causing inconvenience and delay. It should be borne in mind when the snare is used that the wire requires less current to heat it than do the platinum points, and that the thinner the wire the less current required. If any readjustment be necessary the current from the battery should be cut off before the wires are touched by the fingers.

When the instrument has thus been tested the terminal is to be applied cold to the desired point, and the current closed by means of the key on the handle. It must be removed from contact with the tissue while still hot, or it will stick, and produce tearing and bleeding in removal. The connections of the points are easily melted if the current be kept closed too long. After removal from the patient the point should be cleaned by heating it to burn off adhering tissue.

With regard to *after-treatment*, it is important to allow sloughs to fall off before any further application of the cautery is made. An interval of a week or two should therefore elapse between the operations in cases where several applications require to be made. During this time the patient must be exposed as little as possible to septic influences, and this may be aided by the use of antiseptic ointments, sprays, or lotions applied to the affected part.

Galvaniser's Eczema. *See* Dermatitis Traumatica et Venenata (*Chronic Dermatitis*).

Galvanism. *See* Aorta, Thoracic, Aneurysm (*Treatment*); Athetosis (*Treatment*); Electricity (*Galvanism*).

Galvanotaxis.—The movements in cell protoplasm caused by electricity. *See* Physiology, The Cell (*Protoplasm*).

Galvano-Therapeutics.—The use of galvanism (electricity developed by chemical action) for the cure of disease or for the relief of pain. *See* Electricity.

Galvanotonus.—A sustained contraction of muscle which may occur while strong currents are flowing through it. *See* Physiology, Tissues (*Muscle, Contraction*).

Gamboge.—A gum-resin derived from *Garcinia Hanburii.* It is imported in the form of reddish-yellow cylindrical masses. It contains (1) *Gambogic Acid*, a bright yellow resin; (2) *Gum. Dose*—$\frac{1}{2}$-2 grs. *Preparation*—Pilula Cambogiæ Composita, containing Barbados Aloes. *Dose*—4-8 grs. Gamboge is a drastic hydragogue purgative. It causes a great deal of irritation of the intestinal mucosa, and its action is accompanied by painful griping. It is seldom prescribed.

Gamboge, Indian.—Cambogia Indica is a gum-resin obtained from *Garcinia morella*; it is official in the Indian and Colonial Addendum (1900) to the British Pharmacopœia of 1898; it is given for the same purposes as Gamboge, and in doses of $\frac{1}{2}$ to 2 grains.

Gametes or **Gametocytes.**—Reproductive cells before fertilisation. *See* Malaria (*Parasitology*).

Gamma, Angle. *See* Angle Gamma; Refraction (*Prisms*); Strabismus (*Apparent*).

Gamma Rays. *See* Beta Rays; Radium. The gamma rays are believed to be identical with Röntgen rays; they are markedly penetrative; and they are not deflected by the action of the magnet.

Gammacismus.—Difficulty in pronouncing the letters *g* and *k*, or complete inability to do so.

Ganga.—The dried flowering tops of *Cannabis sativa*; a synonym of Indian Hemp. *See* Cannabis Indica.

Ganglia, Basal. *See* Brain, Physiology; Physiology, Nervous System, Spinal Cord and Brain (*Cerebrum*).

Ganglia, Spinal. *See* Physiology, Nervous System (*Spinal Nerves*); Spinal Cord, Medical (*Anatomical*).

Ganglion.

See also Bursæ, Injuries and Diseases of (*Bursæ of Palm, Compound Ganglion*); Hand (*Teno-synovitis*); Wrist-Joint, Diseases (*Tubercular Teno-synovitis*).

Definition.—The word ganglion denotes a localised swelling in connection with a tendon sheath. It is derived from *γάγγλιον*, a hard swelling. Such a swelling is primarily cystic in nature, though it may happen that the cavity of the cyst becomes so encroached upon by the fibrous tissue of the wall as to render the tumour almost completely solid.

Causation.—There is still a considerable diversity of opinion as to the exact etiology of cysts in relation to, or in connection with tendon sheaths. They may arise from—(1) The hernial protrusion of the synovial membrane of the sheath through the outer fibrous tunic, (2) a colloid degeneration of the cells of the synovial fringes, (3) the dilatation of the sub-synovial crypts or follicles of the tendon sheath or adjacent joint cavities, (4) a chronic teno-synovitis, and possibly (5) tuberculous affection of the synovial membrane of the tendon sheath.

There are other cystic swellings that may occur in the same regions as those that are the common sites of ganglia, which, although closely resembling true ganglia, have their origin quite apart from a tendon sheath. Among these may be mentioned bursæ, hygromata, and hydatid cysts.

True ganglia are present more frequently in the female than in the male sex, and are most prevalent from ten to thirty years of age.

Certain occupations and pursuits are apt to be predisposing causes. Musicians, laundry workers, and others who exercise greatly the muscles of the fingers, are peculiarly likely to be affected.

Situation.—Ganglia are most frequently found associated with the tendons of the extensor and flexor muscles of the wrist, fingers, ankle, and toes, and are particularly liable to affect the tendons of the extensor communis digitorum and the extensor indicis, though the extensors of the thumb are almost equally involved. All of these tendons, it will be observed, are on the radial side of the wrist. On the dorsum of the foot the tibialis anticus and the extensor proprius hallucis are the tendons that are most commonly the source of origin. Occasionally ganglia may arise about the insertion of the hamstrings, and still more rarely about the elbow.

Pathological Anatomy.—The tumour is covered, at any rate primarily, with healthy skin, to which it is not adherent. As a rule there is a fibrous cyst wall varying in its thickness. This is more or less perfectly lined by endothelial cells, capable of secreting a characteristic fluid. The colloid material found within the sac possesses the consistence of honey that has been strained, and is usually colourless and alkaline in reaction. It is distinctly proper to ganglia, not being found in any other cavity, being as characteristic of them as the contents of a sebaceous or of a dermoid cyst are for such. It is altogether different from bursal or ordinary synovial fluid.

Ganglionic cysts are attached in nearly every case, as can be demonstrated by careful dissection, to one or more of the neighbouring tendon sheaths, and in addition to the capsule of the joint which is adjacent to them. Intercommunication, however, between the synovial cavity of the tendon sheath or of the joint and the interior of the ganglion is the exception rather than the rule.

Signs and Symptoms.—*Signs.*—Ganglia occur as prominent swellings, varying much in size, but seldom attaining a greater magnitude than that of a walnut. Sometimes they are distinctly globular or hemispherical in shape, and at others flattened. To the touch they are generally densely hard, hence the origin of the term "ganglion," but they may be of a softer character, and they may fluctuate readily, while, when tense, and owing to the nature of the contained fluid, fluctuation cannot always be obtained, yet they are in every instance truly elastic. When present on the palmar aspect of the fingers in connection with their flexor tendons, they may be so firm as to be mistaken for solid tumours. They may become apparently diminished in size according to the position assumed by the joint near which they lie. When the wrist is their situation they will be more tense and prominent when this joint is flexed ventrally, and seemingly almost disappear when it is again fully extended. With the motions of the fingers or toes there may be some movements communicated to the ganglionic swellings.

Symptoms.—The possessor of a ganglion usually complains of the deformity caused by the swelling, and attributes pain to it especially on putting into action the muscles upon the tendons of which it appears to be. This pain may be so severe as to prevent the sufferer from firmly grasping an object in the hand, or of walking in boots with comfort. A sense of weakness of the part is almost always present. Occasionally the actual pain is referred to some distance up the limb. In hysterical or neurotic

patients extreme discomfort will result from the condition.

Diagnosis.—(*a*) From other cystic swellings: 1. Bursæ; 2. Aneurysms; 3. Synovial cysts.

(*b*) From solid swellings: 1. Fibromata; 2. Lipomata; 3. Tuberculous growths; 4. Gummata; 5. Osteomata; 6. Chondromata; 7. Sarcomata.

In the diagnosis from bursæ some difficulty may be experienced, for both varieties of swellings may exist in similar regions, and both may be associated with tendons. Bursæ, however, are as a rule over bony points, while ganglia have no connection with such. Bursæ contain typical bursal synovial fluid, while ganglia are filled with their characteristic colloid material. It is usually in the case of a ganglion in relation with the flexor carpi radialis tendon sheath that a possible error in diagnosis can occur. Here there may be communicated pulsation to the swelling, and if there has been a history of injury, a somewhat close resemblance to a traumatic aneurysm presents itself. Moreover, such aneurysms of the radial artery are not by any means rare. The chief point of distinction lies in the fact that if the swelling is displaced a short way from the line of the vessel the pulsation ceases, and that an exploring needle, introduced only in such cases as will be submitted to operation, reveals the contents to be in one case blood and in the other the usual colloid material.

Synovial cysts may be almost impossible of diagnosis unless their contents are seen, when the synovial fluid will not have the characters of the ganglionic. In the majority of instances the fact that fluctuation can be obtained in a ganglion serves to distinguish it from any of the solid swellings enumerated above.

Treatment.—This may be either non-operative or operative.

Non-operative Treatment.—1. Application of the Tincture of Iodine.—For a considerable period the external application of this solution has found favour in the hands of many. It is, however, not only useless for the removal of the swelling, but it does little to mitigate the symptoms that are produced by the ganglion. Possibly the persistent application of a strong solution of iodine may bring about so much inflammation as to cause positive harm. Iodine should therefore be avoided, unless it is employed solely as a *placebo*.

2. Prolonged Pressure.—There is no doubt that in a few instances this method of treatment has resulted in the diminution or complete disappearance of the tumour. It is at the best only an uncertain means of bringing about the cure of the lesion, and an apparent cure may be speedily followed by a refilling of the non-obliterated cyst cavity.

3. Evacuation of the fluid by the bursting of the sac wall by means of a sharp blow, as from the back of a book, or by the pressure of the surgeon's thumbs. This way of treating a ganglion is the one that is probably most frequently used, but there are some weighty objections to its employment. In the first place, it is as a rule merely palliative, the cyst wall healing, and the fluid ere long again distending it. It is a very painful method. The blow struck to rupture the sac may fail to do so owing to the thickness of its wall, or may cause more damage than was intended, including even that of producing a fracture of the bone beneath. On the other hand, it has certain distinct advantages. It is simple, has only a small amount of danger, involves no risk of sepsis, and leaves no scar.

Operative Treatment.—1. Puncture of the sac with a tenotome, and the squeezing of the colloid contents out through the aperture so made. This method may be somewhat more satisfactory than the former in being possibly less often followed by a recurrence. It should be done only after the part has been rendered aseptic, for otherwise most untoward results may ensue, leading in some instances to destruction of the synovial cavities of the adjacent joints and tendon sheaths. Pressure is to be applied by a pad of gauze and firm bandaging after the contents have been evacuated.

2. Removal of the colloid fluid by puncture, and the subsequent injection of the cavity of the cyst with tincture of iodine or other irritating aseptic fluid. This may lead to the obliteration of the cavity by the setting up of an inflammation which will cause the surfaces to become united by plastic exudation. It is not a certain method of cure.

3. Extirpation of the cyst by dissecting it as a whole from the tissues to which it is attached. This is the only surely radical means of removal. It has for its advantages that the whole of the cyst can be dealt with, and that with the assurance that it will never make its appearance again. It is objected to chiefly on account of the scar that will necessarily result, though it must be remembered that this is but a very slight one provided that strict asepsis is maintained. For ganglia in the lower extremity no method of treatment could be more satisfactory, but for those that are found about the wrist, and particularly those on its dorsal surface, it is perhaps not so much in favour. After the recurrence of the swelling when other methods of treatment have in their turn failed, there should be no hesitation in recommending and even urging the radical removal of the cyst by dissection.

Ganglion, Compound Palmar.—Though this term is in common use, the condition is not one that should receive the designation of "ganglion," as it is in reality in the majority of instances a true tuberculous teno-synovitis of the sheaths of the flexor tendons in the palm of the hand.

Gangrene.

See also Aneurysm (*Complications, Ligature, Effects*); Cholera, Epidemic (*Symptoms*); Dermatitis Traumatica et Venenata (*Causal Agents, Physical*); Dermatitis Traumatica et Venenata (*Feigned Eruptions, Spontaneous Gangrene*); Diabetes Mellitus (*Symptoms, Complications, Gangrene*); Fractures (*Complications*); Glanders (*Symptoms in Man*); Hysteria (*Gangrene*); Lungs, Gangrene of; Nephritis (*Chronic, Clinical Features*); Post-Mortem Methods (*Bacteriological Investigations, Acute spreading Gangrene*); Raynaud's Disease; Scrotum and Testicle, Diseases of (*Gangrene of Scrotum*); Stomach and Duodenum, Diseases of (*Morbid Anatomy, Gangrene*); Skin, Bacteriology of (*Gangrene*); Stomatitis (*Cancrum oris*); Toxicology (*Ergotism*); Typhoid Fever (*Complications and Sequelæ, Gangrene*).

By gangrene is meant the death of a part; but inasmuch as *caseation* and fatty degeneration are both forms of tissue death, and are not included under this heading, the term must be admitted to be a somewhat lax one.

The older authors did not recognise that many of the phenomena they described were really due to micro-organisms grafted upon the gangrenous part, and in consequence their descriptions have to be very materially altered. It must be remembered that loss of pulsation, loss of temperature, loss of sensation, function, and colour, are the sole signs of the death of a part, and that if this gangrenous portion, after being cleansed, be kept wrapped in an antiseptic dressing, no further change will occur in it. Bearing this in mind, the lengthy descriptions which we have in our older books appear very absurd.

Dry and moist are two forms usually admitted by all authorities.

Dry Gangrene is generally met with in old people suffering from atheroma. It is simply death with mummification, and in it the gradual contraction of the calibre of the terminal arteries and the absence of moisture render the tissues a very unsuitable soil for the growth of micro-organisms. Hence septic changes rarely affect the dead part, but the neighbouring living tissues may be the seat of severe septic changes.

The part assumes, instead of a purple dark colour, a tallowy white colour, mottled here and there with brownish streaks due to disintegrated hæmoglobin. The dead tissues become brown, then black, the whole gradually getting mummified.

Moist gangrene is for the most part caused by some obstruction of the venous rather than of the arterial system, so that a complete stasis in the capillaries takes place. If the cause is on the arterial side it is due to *acute* arterial thrombosis, and in the moist, unlike the dry form, there is no previous narrowing of the arteries.

It must be clearly understood that what is generally described as the third stage, or stage of putrefaction, does not exist as part of the gangrenous process, but is due to the presence of micro-organisms in the skin. If the skin be thoroughly disinfected immediately before its death the part will remain aseptic. Thus we recognise two forms of moist gangrene.

(*a*) *Aseptic Moist Gangrene.*—The gangrenous portion, although black, green, or yellowish white, does not change its *size*, and is soon thrown off or absorbed without much disturbance to the surrounding tissues. A good example of this condition is seen in a strangulated aseptic piece of omentum.

(*b*) *Septic or Putrid Moist Gangrene.*—Decomposition is the chief feature in this form of gangrene. The part becomes swollen and boggy, and gives when pressed an emphysematous feeling. Sulphuretted hydrogen, ammonia, and other gases are formed, and give rise to bullæ beneath the cuticle.

The *symptoms* of many cases of gangrene, as described in older works, must be much altered when we recognise that the great majority of the symptoms then described were due really to the absorption of the products of micro-organisms. Practically the symptoms resolve themselves into those produced by a constitutional condition prior to the onset of the gangrene plus the local absorption of ptomaines, or septic poisoning.

These constitutional conditions are most frequently some form of *malnutrition*, *renal disease*, or *diabetes*.

Varieties of Gangrene

(i.) *Traumatic*, or that due to *injury* of the vessels or tissues. It may be divided into two forms—one *direct*, where, so to speak, the

life is crushed out of a part; and *indirect*, where the gangrene occurs at some distance from the seat of the vessel injured. When a part is so severely contused that it is likely soon to be gangrenous, it becomes cold, insensible, and soon its function is interfered with. At the same time there is the change of colour already described. Most forms of direct traumatic gangrene assume the moist characters. They are generally aseptic or become so, because it is so difficult to cleanse the parts thoroughly, and carters and that class are those in whom it is most often seen.

Direct gangrene must not be confused with acute inflammatory or spreading traumatic gangrene, which has special features due to a special micro-organism, but the term must be applied to those cases where so much injury is inflicted on the vessels and tissues that death occurs.

The chief causes of indirect traumatic gangrene are:—(*a*) *Injuries to the main artery and vein.* This is seen frequently in the case of the popliteal or lower femoral arteries, where a compound fracture tears both vein and artery; or it may be that blood is effused to such an extent that it presses on both main artery and main vein and so obstructs the flow of blood within them. (*b*) *Ligature of a main artery* in some people will cause gangrene. (*c*) *Thrombosis* similarly may cause gangrene. If occurring after an injury to a main artery at some distance from where the gangrene exists, it would come under this heading.

(ii.) *Senile gangrene* is one of the commonest forms of gangrene met with. It arises generally from slight injuries to the feet of old people. The calibre of the arteries of the aged becomes very much curtailed from atheroma, and added to this there may be varicose veins or some chronic bronchitis and emphysema. In such a patient anything that upsets the circulation and tends to clotting of blood, either in the large or small arteries, will cause gangrene of toes or foot. The change occurs sometimes in the nose, hands, or tongue, as well as in the lower extremity. The writer's experience is that in the large proportion of cases of senile gangrene there will be found a small abrasion from the boot or from the cutting of a corn, which admits micro-organisms into the small veins, and these easily become thrombosed with a thrombus composed of fibrine and micrococci. This agrees with the descriptions we find of pain, and afterwards a reddish blush, which almost always precede the actual gangrene.

The further history of a case of senile gangrene is, that the patient gets ulcerations, bedsores, a low form of septicæmia, or dies of some complication, such as pneumonia, bronchitis, or exhaustion.

Chemical Changes.—The tissues become oxidised in such a way that carbonic acid, ammonia, and water are the results. Valerianic, butyric, and carbolic acids are formed together with indol, skatol, etc. The gangrenous odour is thus produced. Leucin, tyrosine, margarine, and ammonio-magnesium-phosphate are also formed.

(iii.) *Gangrene due to sudden Obstruction of the Blood-vessels.*—This form may be due to pressure from *without*, as in tight bandaging, pressure of a fracture, or where a plaster of Paris bandage has been badly applied, head of a dislocated bone, or to the application of a ligature. It may also be due to obstruction *within* the vessel (embolic). In the former the limb is at first cold, it is pale in colour, and there is tingling in the peripheral nerves; then the parts get deeper in colour, and soon become purple. Sensation is lost, and the case usually assumes the moist form.

The embolus in the embolic form is usually a fibrinous vegetation, or an atheromatous plate. Neither in the case of embolus nor ligature should gangrene occur if the collateral circulation is good, but there is often present some general condition predisposing to a weak collateral circulation, such as endocarditis in rheumatic fever, or the weak heart of continued fevers.

The pain in *embolic* gangrene is generally felt at that portion of the artery where the embolus has become impacted. This is frequently at the bifurcation of a main artery into its two divisions, as in the popliteal or brachial arteries. A word of advice must be given about the danger of putting severely contused fractures in plaster of Paris, and sending the patients to their homes. This is a practice which is much in use in the German hospitals, but the author has had two patients sent to him by medical men who had done this injudiciously, and gangrene resulted in both cases.

(iv.) *Diabetic Gangrene.*—This comes under the head of gangrene due to general causes. It may be moist or dry, is usually the former, and is due to an endarteritis and peripheral neuritis. The forerunner of grape sugar, or grape sugar itself, causes irritation of the lining of the arterioles, and a thickening and narrowing of their calibre results. Micro-organisms grow very readily in diabetic patients, as is seen in the great tendency diabetics have to form carbuncles.

Diabetic gangrene is as a rule more acute in its course; there is more sloughing and inflammation than in the other forms.

(v.) *Gangrene from ergot* may occur in epidemics, and is due to rye being infected with *claviceps purpurea.* It is preceded often by intestinal symptoms, such as sickness and diarrhœa, and afterwards by coldness and anæsthesia of the limbs. There are two causes of this anæsthesia and coldness—one is contraction of the arterioles, the other a peripheral neuritis. It is considered by some that the

neuritis has more to do with the disease than the contraction of the vessels. Most common in men between thirty and forty, the gangrene of ergot generally is of the dry variety, and may be very slight or involve a whole limb.

(vi.) *Symmetrical gangrene* or Raynaud's disease, as its name implies, affects both sides of the body, thus differing from senile gangrene. Again, the calibre of the arteries in this disease is not contracted. The disease essentially is either a peripheral neuritis or some deep-seated lesion of the spinal cord. It has been started by sudden mental shock or fright, and generally occurs in women.

Maurice Raynaud published his thesis in 1862, and his final contribution in 1874. He described a *local syncope*, a *local asphyxia*, and *symmetrical gangrene*, as three successive stages of this disease.

Local Syncope.—Raynaud was inclined to lay stress on the neurotic origin of this disease. Complete pallor of the two hands occurs, or it may be in the toes, one finger or one toe being affected. The principal difficulty is the performance of slight movements, and often no discomfort is complained of. There is analgesia, tactile sense is present, and changes of temperature can be appreciated.

The attack may last a few minutes or some hours. It is brought about by slight changes of temperature, such as a cold day in the middle of one or two warm ones, or it may occur periodically after the bath.

Neurotics and neurasthenics are the classes in whom it is met with most frequently, and there may be a periodicity in the attack, as after the morning bath.

Slighter causes even than this may produce an attack, such as passing from a warm into a colder room. The mild cases do not tend to go on to the later forms of Raynaud's disease.

Local Asphyxia.—Either the whole hand or foot on each side of the body becomes purplish black, or the attack is limited to one or two fingers or one or two toes on each side.

There appears to be nothing but venous blood circulating in the part affected. Some swelling and a burning or shooting pain accompany the attack, which may last for an hour or two, may often recur, and need not necessarily go on to gangrene. The subsidence of the attack may be sudden or gradual. The more severe cycles last from two to ten months. In addition to the fingers and toes which are most commonly affected, patches may occur on other parts, as about the knees or on the helix of the ear.

If the asphyxia persist, then we get the final stage (*symmetrical gangrene*) of Raynaud. The pain in and darkness of the parts increase, and bullæ may form. There appears to be for a time death of a large area, but this settles down to a more limited portion, as the matrix of a nail or end of a finger. The affection may be simultaneous, or successive parts may become gangrenous. The association of Raynaud's disease with *hæmoglobinuria* must not be forgotten, as many cases of the two diseases running side by side have been reported.

The original hypothesis of Raynaud still seems to hold good as the best explanation of the pathology of this disease, viz. "That vaso-motor centre or centres are unduly irritable, that the commonest irritant is from the periphery, as for example cold, and that the efferent impulses from the centre lead to the paroxysmal contraction of arterioles."

(vii.) *Gangrene due to Heat or Cold.*—(*a*) *Frost-bite.*—When a part is exposed to cold the cutaneous capillaries contract, and the part becomes pale and numb, and soon the tissues assume a wax-like appearance. The further results are usually indirect from secondary inflammation.

The symptoms are numbness, tingling, loss of power and sense of weight, generally in the hand or foot. The limb is at first contracted, and shortly afterwards becomes swollen. It is not unusual for parts to look well for some days and then become bluish, dark blue, and finally black. Subsequently a line of demarcation forms, ulceration takes place, and either a toe or a finger, or possibly a foot or hand, is lost. The ulceration may amount simply to a small patch of skin, say about the os calcis or on the outer side of the foot.

Morbid Anatomy.—Cold has at first a stimulating effect upon the circulation; but if it be severe or long continued, then the blood in the smaller veins becomes frozen, and the skin assumes very much the appearance that is seen after the use of the ether spray.

The blood is absolutely frozen and there is complete stasis, and when thawing takes place, so much damage has been done to the vessel-wall itself that it permits the blood to pass through, and hence we get those inflammatory changes which are the real cause of gangrene in frost-bite.

A still longer exposure and the involvement of a greater extent of surface induces the collection of blood in the larger veins, and thus the internal organs become œdematous, and there is fatal stupor, coma, and asphyxia.

The danger from frost-bite is in proportion to the length of time and extent of surface exposed, and to the power of resistance of the individual. It must be remembered that alcoholics are very liable to frost-bite.

Dry cold is said to have a more severe effect than moist cold, and if a wind is blowing the effect will be much more severe. The author had under his care seventeen patients from a crew shipwrecked on the Newfoundland coast. An important observation was made by the men, namely, that those who had not removed the water out of their sea-boots had escaped, while

all those who had became affected with gangrene. They were in the open for sixteen days, and the presumption was that the water was heated by the body and kept the feet from freezing.

Cases are related where persons have been buried in *dry* snow for many days without much food and recovery has taken place, but if the snow has been thawing, a very much more serious effect has been produced.

This is singularly well pointed out by Larrey, the distinguished French surgeon, who, in the Russian campaign, found that though none of the soldiers suffered from many days' exposure to *dry* cold and frost, as soon as a thaw came a large number of both guard and line became frost-bitten.

Chilblain is a less serious local effect of cold, as are chaps, fissures, and frost erythema (pernio). The lips, the ears, the fingers, and toes are the portions of the body most frequently affected. Young girls of lymphatic constitution or those who inherit an irritable nervous system are most liable to this complaint.

Sudden changes of temperature are generally attributed as causes, but many people get chilblains who never leave the house in cold weather. As in frost-bite, so in this disease, damp cold, as in a sudden thaw, is a much more frequent exciting cause than dry cold.

An eruption of dull red patches, not well defined, raised, tumefied, and burning, may be followed by vesicles or bullæ. Subsequently an ulcer excavated and with thick edges may form. A peculiar viscid discharge is often present. Three degrees have been described: (1) simple congestion with itching and extreme tenderness; (2) vesication; and (3) death or sloughing with or without marked erythema.

Chilblain is merely an early stage of frost-bite, according to some authorities, but there is little doubt that in most cases of chilblain the nervous system, as in Raynaud's disease, plays an important part.

(*b*) *Burns.*—It is in the last three degrees of burns and scalds that the effects produce gangrene. In these three we find that sloughs have to be removed before the healing process can take place. These effects will depend on the source, duration, and intensity of the heat.

(viii.) *Gangrene due to the use of carbolic acid* is met with under dressings of this acid. It is of the dry variety, and affects the fingers and toes for the most part. Apparently it is more often met with after the prolonged application of weak solutions, and is not so often seen after the application of *pure* carbolic acid.

A weak terminal circulation should contra-indicate the use of carbolic acid dressings. The American authorities speak of as many as one case of carbolic gangrene in 1000 surgical cases, but this is certainly not the experience in this country. Personally I have seen only one case of gangrene that could be certainly traced to the use of carbolic acid.

Multiple gangrene of the skin occurs independent of the use of carbolic acid. Blebs occur in many parts, and afterwards numerous small particles of skin die. The nervous system is supposed to play an important part in this disease.

(ix.) *Infective gangrene*, or the gangrene due to the presence of micro-organisms.—This is a most important heading, and one which includes such diseases as glanders, plague, beri-beri, hospital and acute spreading traumatic gangrene, etc.

(*a*) *Spreading traumatic or acute traumatic gangrene* is an extremely rapid and very often fatal form, due to a micro-organism, the bacillus of malignant œdema, first isolated by Koch. It is closely allied to symptomatic anthrax in cattle. The bacillus of malignant œdema is rod-shaped, somewhat like anthrax, but more slender. It is capable, even in a test-tube, of forming hydrogen and carburetted hydrogen. It grows very readily in musk, and is found in garden earth.

The course of the disease is very rapid. It, as a rule, appears from two to three days after a severe accident, generally a compound fracture or a compound dislocation, but it may occur after a small inoculation. It is usually fatal in three or four days if very active measures are not adopted. As a general rule it is predisposed to by drunkenness which has been long continued, as for instance in barmen and brewers' draymen.

The limb becomes hard, tense, white, and œdematous, but soon goes on to a dusky red and then livid colour. There is gas, chiefly hydrogen and carburetted hydrogen, evolved, and crackling is present; at the same time the constitutional symptoms are very severe, and delirium soon supervenes.

(*b*) *Hospital gangrene* and wound phagedæna are now very rarely seen. I have never seen a case, but before hospital wards were so well ventilated and antiseptic precautions taken it was a common enough disease. A pseudo-membranous slough forms within eight hours to three or four days of the infliction of a wound; the granulations, if present, become ashy grey. The surface of the wound is dry and the edges pale. Then either an ulcerative or gangrenous process follows. In the gangrenous form the disease is often fatal in from twenty-four to forty-eight hours.

(*c*) *Cancrum oris or Noma.*—Under this heading we have to deal with a very fatal disease which occurs in children from two to seven years of age, and in most cases after measles or scarlatina, especially the former. On the gum or immediately inside the cheek an ulcer occurs, which at first is slimy and white, but soon becomes gangrenous; a black spot appears on the outside of the cheek and rapidly spreads, so that the

side of the face may be destroyed, the bones occasionally being involved. In other cases the process is a gangrenous stomatitis, giving rise to a foul breath from fœtid material which has been swallowed. The symptoms of acute poisoning (sapræmia) soon show themselves, and the patient becomes comatose and dies in three or four days. Septic pneumonia is often a cause of death in this disease. (*See* "Stomatitis.")

Bacteriologically a long delicate bacillus has been proved to be the cause. The author has had under his care two cases of noma which recovered after the free use of the thermo-cautery, and in which extensive plastic operations had subsequently to be performed. The word noma is sometimes restricted to this process occurring about the genitals of children.

(*d*) *Carbuncle and boil and acute necrosis* are classed by some authors as forms of gangrene.

(*e*) *Gangrene in beriberi* has been seen. A thin black line crossed the tips of the toes, and dry gangrene followed in both feet, requiring amputation of both legs at the seat of election. Undoubtedly the gangrene was due to a peripheral neuritis which so often occurs in this disease. The neuritis is caused by the direct action of toxins (produced by a micro-organism) on the nerve itself.

Treatment.—Some general remarks will be made on the treatment of gangrene, and then the treatment of each variety will be dealt with shortly.

To begin with, the whole treatment of gangrene must be altered now, our methods have so much improved, and especially is this true as regards the question of amputation. We can, with safety and with much less chance of sloughing of flaps, amputate very much earlier than we formerly could, thus saving the patient much painful suffering and danger from the presence of a septic dead mass of tissue. A second important result of our better antiseptic methods is the fact that we can boldly cut down on a ruptured aneurysm or compound fracture, relieve pressure on vessels of effused blood, and thus often prevent gangrene. It is most important in all cases of gangrene to get at the exact cause; thus a tight bandage should be removed at once, or a free incision should be made in inflammatory tissue, if causing obstruction to the flow in the main vessels. The stricture in a piece of strangulated bowel or omentum should be divided at once, and so with a paraphymosis, if elevation and attempts at reduction fail.

If in a limb the main vessels cannot be relieved, then the limb should be raised, the joints flexed, and the parts should be lightly wrapped in cotton wool.

If the gangrenous part has not become septic, then the greatest care must be taken to cleanse skin, nails, etc., and salicylic wool and iodoform should be placed between the toes or similar situations after thorough cleansing. If septic, such a limb should not be poulticed, but a boracic fomentation should be placed a little above the line of demarcation.

Sloughs have to be removed, and the best application is a bran poultice covered with boracic powder.

Wait for a line of demarcation, keep aseptic if possible, but don't wait too long, especially in septic or spreading cases, are the broad rules which must guide our judgment.

A nourishing diet, stimulants, and tonics are also required, and small doses of opium, except where there is diabetes or renal disease.

Where these constitutional diseases exist, special dietetic and medicinal treatment must be employed.

Traumatic.—Warm antiseptic lotions must be used freely to thoroughly cleanse. Ascertain if there is any pulsation; if not, see that the main vessels are not pressed on by extravasated blood or compound fracture. Do not amputate at once if there is much shock, but rather wait for eight or ten hours. I quite agree, from a large experience of severe smashes, with those authorities who advocate waiting in order to let the patient recover and the parts be made aseptic. The rule for amputating is, to do so, only when you are convinced that there is no circulating blood in the tissues.

On the other hand, we must not try to save the part in the case of albuminuria, diabetes, or other constitutional disease.

The subject of these diseases will not stand the long-continued drain upon them, and very often amputation has to be resorted to later, when strength to rally no longer remains.

Raynaud's Disease.—Preventive, tonics, frictions, warm douches, electric baths, attend to any uterine complication, keep aseptic. In no case is it necessary to amputate. Rather cover with iodoform and allow tips of fingers to gradually drop off.

Ergot.—In this form it is very rare to find more than a finger or toe affected, and amputation is not generally necessary.

Diabetic.—Codeine $\frac{1}{4}$ to 5 grs., or small doses of opium, very useful. In such cases any septic wound should be specially looked after, as its neglect is highly dangerous.

Amputate very early; rule here completely changed from what it used to be. If there is a plug high in a main vessel, don't do a regular amputation, but dust with iodoform, divide bone, and allow to drop off gradually.

Senile Gangrene.—Early amputation is now the treatment, well away from the gangrene, and in the case of the foot through the knee-joint, even if pulsation be felt below. The patient here must be warned about septic abrasions and wounds about the toes.

Frost-bite.—Prevent, by warm clothes and absence of moisture. Patient should eat much fat and avoid alcohol. When actually frozen, the part should be most carefully thawed with cold or snow water and gentle friction. Gradually raise temperature around patient. Elevate if œdema.

Render, if gangrenous, aseptic with dry applications. Amputate early. For the milder forms, as in chilblain, the same treatment must be adopted, and painting with fairly strong solutions of carbolic acid, or belladonna and collodion, or the old-fashioned brandy and salt mixture, are amongst the best remedies.

Acute spreading Gangrene.—The point in this disease is to remember not to wait for a line of demarcation, but to amputate as soon as possible. The shoulder or hip operation must be chosen, and care must be taken not to soil the flaps. Stimulants, incisions, and baths will be necessary. It is a most fatal disease; about 5 per cent recover.

Noma. — If in mouth, antiseptic washes, sanitas, and boro-glycerine. Then scrape with Volkmann's spoon, or destroy with actual cautery or pure carbolic acid, remove dead bone. If recovery take place, Thiersch-Grafting or plastic operations will be required. Iron, quinine, and stimulants will be necessary.

If in genitals, then baths of sanitas or boracic acid, and powder with iodoform.

Gannister Disease.—A morbid condition of the lungs due to the inhalation of particles of dust, common in stone masons, especially "gannister" workers; silicosis or pneumonoconiosis.

Ganser's Syndrome.—A symptom-complex or combination of symptoms, including disturbance of consciousness, amnesia, hallucinations, senseless answers to questions, and physical disturbances, described by Ganser; it is generally associated with hysteria. It is fully described in Julius Hey's book, *Das Gansersche Symptom*, Berlin, 1904.

Gaol Fever. *See* Typhus Fever (*Etiology*).

Garden Cities.—Garden cities are proposed in the hope of ameliorating the conditions of the working class, who, in towns, are under certain disadvantages; they are to be constructed in the country and are to have the best hygienic arrangements possible. The first Garden City, that at Letchworth, near Hitchin, is now (1906) in progress of construction, and has a population of over 1600. The great difficulty to be anticipated will doubtless be the disinclination of the working man to leave his urban surroundings for such Garden Cities.

Gardiner Browne's Test. *See* Ear, Examination of (*Hearing Tests, Tuning-Fork*); Ear, Middle, Chronic Non-Suppurative Disease (*Diagnosis of Middle from Internal Ear Disease*).

Gardone Riviera. *See* Therapeutics, Health Resorts (*Italian Lakes*).

Garfield's Filter.—A method for the disposal of sewage, consisting of a filter bed of coal, well washed, and 4 or 5 feet deep.

Gargarismus.—The act of gargling.

Garget.—A specific mammitis in the cow. *See* Milk (*Pathological, Diseases of Cows*).

Gargle.—The application of a liquid (containing an astringent, demulcent, or cleansing medicine) to the back of the throat, by taking it into the mouth, throwing the head well back, and forcing air through it by expiration. *See* Pharynx, Chronic Pharyngitis (*Treatment*); Prescribing.

Garlic, Oil of. *See* Allyl; Asafœtida.

Garrod's Thread Test.—The excess of uric acid (in gout) as revealed by the incrustation of a thread suspended in blood serum (obtained from a blister), to which a few drops of acetic acid have been added; the value of the test has been weakened by recent investigations.

Garrulitas Vaginæ.—A disagreeable condition in which gas, often bad-smelling, passes from the vagina; it may be due to relaxation of the vulvar structures, and then atmospheric air is first sucked into the canal and then expelled again, sometimes with an audible sound; in other cases it may be due to fistulous communications with the intestine by which flatus gains access to the vaginal canal; in the former cases a plastic operation on the perineum is required, in the latter the fistula must be closed (if possible).

Gartner, Duct of. *See* Generation, Female Organs of (*Urethral Canal*). The name is often spelt erroneously *Gärtner*.

Gas. *See* Asphyxia (*Causes*); Suppuration (*Gas-producing Micro-organisms in Pus*); Urine, Bacteria in (*Pneumaturia, Gas-forming Organisms*).

Gases. *See* Medicine, Forensic (*Irrespirable Gases*); Stomach and Duodenum, Diseases of (*General Symptomatology, Local Symptoms, Flatulence*).

Gasserectomy.—Excision of the Gasserian ganglion, a somewhat serious operation recommended in the worst cases of trigeminal neuralgia.

Gasserian Ganglion. *See* Herpes (*Pathology, Changes in the Gasserian ganglion in herpes ophthalmicus*); Nerves, Neuralgia (*Trigeminal, Treatment, Excision of the Gasserian Ganglion*).

Gastein. *See* Balneology (*Austria, Indifferent Thermal Waters*); Mineral Waters (*Thermal*).

Gastralgia. *See* Alcohol (*Indications, Neuralgia, Gastralgia*); Gout (*Irregular, Alimentary System*); Hysteria (*Disorders of Digestive Organs*); Stomach and Duodenum, Diseases of (*General Symptomatology, Pain in the Stomach*).

Gastralgokenosis. — Pain in the stomach, occurring in paroxysms, and relieved at once by taking food.

Gastrectasis. — Dilatation of the stomach.

Gastrectomy. — Resection of the stomach; the excision of the whole or of part of the stomach. *See* Stomach, Surgical (*Operations for Malignant Disease*).

Gastric.—Relating to the stomach. *See* Astringents (*Uses, Hæmorrhage from Stomach*); Chlorosis (*Gastric Ulcer, etc.*); Colic (*Diagnosis*); Digestion and Metabolism (*Digestion in the Stomach*); Gastro-Intestinal Disorders of Infancy; Hysteria (*Infantile*); Leucocytosis (*Lymphocytosis, Gastric Catarrh*); Malaria (*Symptoms, Gastric Form*); Melæna (*Gastric Ulcer*); Palpitations (*Causes*); Peritoneum, Acute Peritonitis, General (*Etiology*); Pharmacology (*Gastric Sedatives, etc.*); Physiology, Food and Digestion (*Alimentary Canal, Functions, etc.*); Stomach and Duodenum, Diseases of; Stomach, Surgical Affections of; Tabes Dorsalis (*Symptomatology, Gastric Crises*); Typhoid Fever (*Gastric Fever*).

Gastritis. *See* Appetite (*Loss, Gastritis*); Gastro-Intestinal Disorders of Infancy (*Gastritis*); Stomach and Duodenum, Diseases of.

Gastro-Anastomosis.—The operation in which a free communication is established between the two compartments of an hour-glass stomach; gastro-gastrostomy. *See* Stomach, Surgical Affections of (*Operations on the Stomach*).

Gastrocele.—Hernia of the stomach.

Gastrocnemius. *See* Muscles, Traumatic Affections of.

Gastrocolostomy.—The operation by which a permanent communication is established between the stomach and the colon.

Gastro-Diaphanoscope. *See* Enteroptosis (*Diagnosis, Einhorn's Gastro-diaphanoscope*).

Gastrodidymus. — United twins, in which the abdominal cavity is single and common.

Gastrodiscus Hominis.—An amphistome or fluke which is not infrequently met with in India as a cause of intestinal distomiasis.

Gastroduodenitis. — Inflammation affecting both stomach and duodenum.

Gastrodynia. *See* Stomach and Duodenum, Diseases of (*Special Symptomatology, Sensory Neuroses*).

Gastroectasis (*or Gastrectasis*). *See* Stomach and Duodenum, Diseases of (*Morbid Anatomy, Malformations and Malpositions*); Stomach and Duodenum, Diseases of (*Special Symptomatology of Dilatation of the Stomach*).

Gastroenteralgia.—Pain, especially neuralgic, of the stomach and intestines.

Gastro-enteritis. *See* Alcohol (*Uses*); Gastro-Intestinal Disorders of Infancy (*Diarrhœa*); Snake-Bites and Poisonous Fishes (*Symptoms of Poisoning from Fish*); Tetany (*Groups of Cases, Gastro-Intestinal*); Typhoid Fever (*Diagnosis*).

Gastro-Enterostomy. *See* Stomach, Surgical Affections (*Operation for Gastric Ulcer, Hour-glass Stomach*); Stomach, Surgical Affections (*Operations on the Stomach, Anterior and Posterior Gastro-Enterostomy*).

Gastro-gastrostomy. *See* Gastro-Anastomosis.

Gastrohysterorrhaphy. Hysteropexy (*q.v.*).

Gastro-Intestinal Disorders of Infancy.

See also Atrophy, Infantile; Children, Development of (*Stomach and Intestine*); Cholera Nostras; Convulsions, Infantile; Heart, Myocardium and Endocardium (*Effects of Cardiac Disease*); Infant Feeding; etc.

Congenital Anomalies

I. *The Œsophagus.*—Congenital anomalies occur more rarely here than at any other part of the alimentary tract. The following conditions have been found:—

(1) Transposition.—In association with transposition of the stomach the œsophagus passes down on the right side of the vertebræ.

(2) Absence or Obliteration, partial or complete.—One variety of this shows very constant characteristics: the pharynx with the upper $\frac{3}{4}$ inch of œsophagus is dilated, and forms a blind pouch ending just below the level of the cricoid cartilage; below this the upper part of the œsophagus is absent, or represented only by a band of fibrous tissue, or a few muscular fibres; the lower part is normal, and opens normally at the cardiac orifice, but above it communicates, by an opening about $\frac{1}{10}$ inch in diameter, with the trachea $\frac{1}{2}$-$\frac{3}{4}$ inch above the bifurcation. Such a condition may be suspected during life, when all food is regurgitated through the nose and mouth almost directly it is taken, and a sound cannot be passed down the pharynx more than about 5 inches from the lips. Death occurs from starvation in a few days; one case at Great Ormond Street lived fourteen days with rectal feeding.

The most satisfactory explanation of this condition is that suggested by Mr. Shattock (*Path. Soc. Trans.* 1890), who assumes that, at the time of the budding of the diverticulum from the anterior wall of the mesenteron to form the lower part of the trachea and the lungs, a kinking forward of the posterior wall occurs, so that the lumen of the mesenteron, which should persist as the œsophagus, is occluded just above the level where the tracheal diverticulum is given off. The segment of the mesenteron below the kink, subsequently the lower part of the œsophagus, remains in communication with the tracheal diverticulum. "On the subsequent formation of the upper portion of the trachea the whole of the unduly contracted lumen (of the mesenteron) immediately above the diverticulum becomes involved in the formation of the air-passage."

(3) Stenosis.—This occurs most often at the upper part of the œsophagus, about the level of the lower margin of the cricoid cartilage, more rarely at the lower end just above the cardiac orifice. This condition may be recognisable during life by the difficulty in swallowing. It has been treated by the passage of bougies.

(4) Diverticula.—It seems doubtful whether any diverticula from the œsophagus are actually congenital. A shallow pit is sometimes found in quite young children at the level of the bifurcation gland, where this has been caseous, but this is probably the result of traction rather than a congenital anomaly. The congenital diverticula which result from persistence of branchial clefts communicate with the pharynx, not with the œsophagus.

II. *The Stomach.*—(1) Abnormal Position.—The stomach may be transposed together with the heart and other viscera. It may project alone, or with part of the transverse colon or small intestine, into either pleural cavity, usually the left, owing to some deficiency of the diaphragm.

(2) Persistence of Fœtal Shape and Position.—The tubular shape and vertical position are sometimes retained. Some authors state that in early infancy some tendency to such a fœtal condition is the rule, and no doubt this is true of the vertical position, but in the writer's experience any tendency to tubular shape has been extremely rare.

(3) Abnormal smallness has occasionally been observed.

(4) Hour-glass contraction or a partial septum may almost divide the stomach into two compartments.

(5) Stenosis or atresia of the cardiac, or less rarely of the pyloric orifice, may be present, and is probably quite distinct from the congenital hypertrophy of the pylorus described below.

III. *The Intestines.*—(1) Abnormalities of position are very common in the large intestine apart from transposition of viscera which may involve the intestines. The sigmoid flexure, relatively much longer in the infant than in the adult, is often much above its average length even for an infant, and may reach over to the right iliac fossa, or form a loop upwards reaching to the right or left costal margin before turning down to the rectum (*vide* Constipation, p. 418). The cæcum also occasionally lies in an abnormal

position, *e.g.* in the mid line at the level of the umbilicus.

(2) Absence or undue Shortness.—This is very rare. In one case the small intestine measured only 2 feet (*Bart. Mus. Cat.*); in another the colon was absent; in another there was no vermiform appendix.

(3) Stenosis or Obliteration.—This is found occasionally in any part of the intestine, but much more often in the small than in the large bowel. Of cases occurring in the small intestine, 42 per cent are in the duodenum (Silbermann). Where multiple stenoses occur it seems likely that a volvulus during intra-uterine life may have caused them. In some cases fœtal peritonitis has been present, and seems a more likely cause. The bowel above the stenosis is usually much dilated, while that below is small, or for some distance may be represented only by a fibrous cord. The condition can only be suspected during life from symptoms of intestinal obstruction, where these are not due to imperforate rectum or anus. Complete occlusion generally proves fatal in a few days; cases of slight stenosis have lived to adult life.

(4) Diverticula.—The persistence of part of the omphalo-mesenteric duct not uncommonly causes a diverticulum in the ileum 2-3½ feet above the ileo-cæcal valve, usually as a blind pouch ½-3 inches in length. The writer found such a diverticulum eleven times in 500 autopsies. Sometimes from the end of the diverticulum a fibrous cord, representing the obliterated portion of the duct, passes to be attached at the umbilicus, or the whole duct may be patent up to the umbilicus, forming a fæcal fistula from which prolapsed mucous membrane often protrudes. In either case the persistent duct serves as a fixed band under or over which coils of intestine may at any time become strangulated. Fæcal impaction may also cause inflammation in the blind diverticulum, and not very rarely the diverticulum is found forming part of an external hernia. Diverticula elsewhere are very rare, especially in the large intestine.

(5) Imperforate Anus or Atresia Recti.—This is due to imperfect development of the hind gut and its failure to meet the proctodæal invagination. It occurs in various degrees: (*a*) there may be only a thin septum separating a well-marked anal depression from the blind end of the gut; (*b*) the rectum may end blindly 2-3 inches away from the surface, and there may be little or no trace of an anal depression; (*c*) there may be a septum occluding the rectum 1-3 inches above the anus, although below the occlusion the rectum and anus are normal; (*d*) the rectum may open into the urethra, bladder, vagina, or even in the loin (*vide* "Diseases of the Rectum").

(6) Rectal or Anal Stenosis.—Without being actually imperforate the anal opening is sometimes abnormally small (p. 113). In such cases it may be stretched under an anæsthetic or enlarged by incision. Similarly the rectum may show narrowing of its lumen or a partial septum at any part of its course, and dilatation or incision may be necessary.

Diseases of the Œsophagus

Acute œsophagitis, as a primary condition, is occasionally found in early life, chiefly during the first year. It is scarcely recognisable clinically. The symptoms are unwillingness to suck, pain on attempting to swallow, and regurgitation of food almost directly it is taken. Tenderness on pressure over the lower part of the trachea is also said to be present (Morell Mackenzie). As a secondary condition œsophagitis has been found with the specific fevers, and the writer has met with it in fatal cases of infantile diarrhœa. In such cases it produces no special symptoms. The application of leeches or of hot fomentations to the neck has been recommended in the rare cases where the condition is suspected during life.

Thrush sometimes spreads down from the pharynx into the œsophagus, and forms a milky-white layer which is found to consist of the spores and mycelium of the so-called "oidium albicans" mixed with epithelial cells; the fungus may penetrate deeply between the cells of the mucosa, and has been found forming so thick a layer as even to obstruct the passage of food.

Diphtheria not very rarely causes a membranous œsophagitis. In forty consecutive autopsies on children with diphtheria the writer found membrane in the lower 1-2 inches of the œsophagus in three cases, and intense congestion of the same area in two others.

Ulceration of the œsophagus is very rare. Superficial erosions have been found as part of the acute œsophagitis of infants, and rarely in older children. The pocks of variola have been found in the œsophagus in children as in adults (Steffen). The least rare variety is tuberculous ulceration from without; the caseous bifurcation gland ulcerates through into the œsophagus about two inches above the cardiac orifice. The writer found such a perforation four times in 270 autopsies on tuberculous children. In none of these cases were any symptoms produced, but hæmatemesis and melæna have occurred. Tuberculous ulceration apart from such direct extension is extremely rare. The writer met with it twice in the same series of cases.

Softening of the œsophagus, like the similar condition in the stomach, with which indeed it is usually associated, is probably an entirely post-mortem phenomenon. It seems to be much commoner in infancy and early childhood than in adults, and it is remarkable that it is much more often found with intracranial disease than with any other condition: the writer met with it eight times in 500 autopsies in children; seven were cases of tubercular meningitis, the remaining one was a meningocele. The lower end of the œsophagus, about 1 inch above the

cardiac opening, is almost diffluent, and in the middle of the softened area there is generally a perforation with thin, frayed edges. The connective tissue of the posterior mediastinum has disappeared, and the nerves and vessels are cleanly dissected out by the action of the digestive fluid which has escaped through the perforation. Usually the adjoining parietal pleura on one or both sides is also softened and perforated by digestion, and an ounce or more of opaque reddish brown fluid, sometimes with partially digested food, is found in the pleural cavity. The posterior and lower part of the lung also is softened, greenish brown, and friable.

Diseases of the Stomach and Intestine

Congenital Hypertrophy of the Pylorus.—Since the year 1841, when this condition was first recorded, a considerable number of cases have been observed in which obstruction of the pylorus existed in infants soon after birth, and it seems likely that this condition is less rare than was at first supposed.

Boys seem to be rather more often affected than girls: out of nineteen cases twelve were males, seven females; but the numbers are at present too small to afford reliable information on this point. No family predisposition has been observed.

Symptoms.—The infant is born healthy, and may be a fine child at birth. For the first week or two it takes food well, and there is no vomiting, or only such slight regurgitation as may be natural at this age. This latent period of apparent health varies, in very few cases vomiting has begun on the day of birth, in about half the cases the onset of vomiting has occurred within the first fortnight, in several cases it has been delayed until the fourth, fifth, or even sixth week. Vomiting is the first symptom, and for a time gives rise to but little anxiety, for at this stage it may occur only once or twice in the day. At the same time the bowels are costive. A change of feeding is tried, and the vomiting perhaps diminishes, but after a few days increases again; other changes of diet are tried, but soon cease to have any influence on the vomiting, which becomes more and more frequent until almost every food is vomited. In some cases, especially in the early stage, and when small feeds are given, the food is retained for some hours, and then vomited in large quantity; more often, however, the vomiting occurs a few minutes after a meal, and in the later stage immediately after swallowing. There is usually no retching, and when the condition is advanced the food seems to be pumped up forcibly by the hypertrophied stomach.

The vomit consists of curdled milk or of whatever food has been given, but soon some gastric catarrh occurs, and mucus is mixed with the vomit, and sometimes even streaks of blood. Where there is much dilatation of the stomach the vomit has a frothy appearance. The absence of bile from the vomit is particularly noticeable, as pointing to obstruction of the pylorus. Pain is often absent throughout, but in some cases there seems to be some pain accompanying the act of vomiting.

A very characteristic sign of congenital hypertrophy of the pylorus is visible peristalsis of the stomach; in some cases it is so obvious that it has attracted the notice of the parents, in others it is only noticeable with care, and to elicit it gentle flicking of the abdominal wall may be required. A rounded prominence appears bulging forward in the epigastrium, first just below the left costal margin, and then travelling with a slow vermicular movement across the epigastrium to disappear at the right costal margin. This movement from left to right serves to distinguish it from peristalsis of the colon, in which the movement is from right to left.

An even more important sign of this condition is a palpable tumour in the position of the pylorus. This has been present in several of the cases, but has not been detected usually until several weeks after the onset of vomiting; the earliest date at which it has been felt is the twenty-seventh day after birth (J. Thomson), about three weeks after the onset of vomiting. Even in cases where the pylorus is easily palpable, it is not so at all times alike; its palpability depends on the temporary condition of the muscle, and, like an intussusception, it may be felt only when there is some contraction of its muscular coat occurring. For this reason one examination is not sufficient to exclude the presence of a palpable enlargement of the pylorus.

Associated with the vomiting there is almost always much constipation, and when the vomiting becomes severe there is marked diminution in the amount of urine passed. As less and less of the food is retained, wasting becomes extreme, the temperature becomes subnormal, and in most cases the child dies of exhaustion.

Morbid Anatomy and Pathology.—The hypertrophy of the pylorus is due to an increase in the thickness of its muscular coat, chiefly in the circular layer. In a few cases some increase also of the connective tissue has been found, but this is exceptional. The cause of the muscular hypertrophy or hyperplasia is still undetermined; it seems certain that it is partly an extra-uterine occurrence, although there can be little doubt that some congenital abnormality underlies it. Some would regard the excess of muscle tissue as a developmental error, a congenital malformation; others consider that this excess is simply the manifestation of increased function from some cause, either extra- or intra-uterine. Perhaps the most probable theory is that put forward by Dr. John Thomson, who suggests that some functional disturbance of the

nervous mechanism of the stomach occurs *in utero*, causing antagonistic action of the muscles of the stomach with spasmodic contraction, and so hypertrophy of the pylorus. As a result of the obstruction at the pylorus the muscular wall of the stomach, and sometimes also of the œsophagus, shows considerable hypertrophy, and sometimes there is considerable dilatation of the stomach.

Diagnosis.—A history of persistent vomiting associated with constipation in an infant under six months of age should always arouse a suspicion that there may be congenital hypertrophy of the pylorus, and where the vomiting resists all treatment, and is associated with visible peristalsis of the stomach, and a palpable tumour in the position of the pylorus, the diagnosis is certain. Before these signs have become evident the condition may be mistaken for simple gastric catarrh; this, however, is more often associated with diarrhœa, and the vomiting can usually be referred to some obvious fault in diet, and is more influenced by treatment. It must be remembered that regurgitation of food is quite common and physiological in healthy infants during the first few weeks of life, any over-distension of the stomach being thus provided against.

From congenital stenosis of the pylorus without hypertrophy, diagnosis is impossible unless a palpable enlargement of the pylorus is detected. Stenosis of the upper part of the duodenum may also closely simulate this condition.

Prognosis.—There is increasing evidence to show that congenital hypertrophy of the pylorus is not always fatal, and in one case which recovered and died nearly a year later of broncho-pneumonia, the hypertrophy of the pylorus could still be demonstrated (F. E. Batten). In most cases, however, it proves fatal. In nineteen cases in which the age at death was recorded, the average age was $9\frac{1}{2}$ weeks at death, the earliest was twenty-one days, the latest nearly five months. The interval between the onset of symptoms and the fatal ending varies from eighteen days up to twelve weeks.

Treatment.—The most valuable method of treatment is feeding by stomach-tube through the nose or mouth. In several cases this has produced considerable temporary improvement, and in one case a spontaneous recovery seemed to follow the improvement started by nasal feeding. Drugs are of some value when gastric catarrh is present as a complication; in such cases sodium bicarbonate, with a minute dose of aqua laurocerasi, may be useful, or the stomach may be washed out with a weak alkaline solution. Where dilatation is considerable and the food collects in the stomach, this should be washed out daily. Careful dieting must not be neglected, for the vomiting is certainly influenced to some extent by the food; it is important, therefore, to select such food as is likely to prove least irritating to the stomach; for this purpose it may be wise to peptonise the milk, or to use whey with or without cream (*vide* "Infant Feeding").

The question of surgical treatment naturally arises, but at present there is not sufficient experience to show how far it may be of value. It has been tried at least three times—twice with rapidly fatal result, once with a favourable result at the age of eight weeks (Abel). It is obvious that such a severe operation as gastro-enterostomy, or any of the procedures which would be necessary to relieve the obstruction, cannot be otherwise than extremely risky at so tender an age and in so feeble a state. For the diagnosis can hardly be made with certainty until some weeks have elapsed since the onset of vomiting, and by that time the infant is in poor condition for such an operation. Moreover, as it seems now quite certain that cases have recovered without operation, it may be said without hesitation that the risk of surgical interference should never be undertaken until a fair trial has been given to medical treatment, especially to the method of feeding by stomach-tube.

Gastritis.—Gastritis is an occasional result of acute dyspepsia in infancy. As the result of irritant poisons it may, of course, occur at any age.

Membranous gastritis is found occasionally in children, usually with diphtheria. The membrane is situated near the cardiac orifice, and is associated sometimes with membrane in the lower part of the œsophagus, and almost always with severe pharyngeal diphtheria. The writer has also found membranous gastritis associated with membranous colitis in an infant with severe broncho-pneumonia where there was no evidence of diphtheria. It has also been seen in tuberculosis.

The condition cannot be recognised during life, but has been associated usually with severe vomiting.

Gastric ulcer is very rare in childhood. It occurs most often in new-born infants probably as a result of congestion owing to the circulatory changes at birth. It is then sometimes solitary, but perhaps more often occurs as multiple erosions. Such ulceration either in the stomach or in the duodenum may give rise to fatal hæmatemesis and melæna; these symptoms occur almost always before the tenth day, usually within the first forty-eight hours of life (*vide* "New-born Infant").

Follicular erosions have been found in severe cases of gastric catarrh in infancy, sometimes also in association with specific fevers, and sometimes with severe burns (S. Fenwick). They very rarely give rise to any special symptoms; severe hæmorrhage has, however, occurred. Perforating gastric ulcer, exactly similar to that

found in adults, has occasionally been found in children; the earliest case was at $2\frac{1}{2}$ years.

Tuberculous ulceration of the stomach is less rare in infancy and early childhood than at any other period. The writer met with it six times in 270 autopsies in tuberculous children; in three of these the tuberculous character was verified by microscopic examination. In all the recorded cases there has been extensive tuberculosis in other parts of the body, often in the intestine and peritoneum. Special symptoms are usually lacking, but fatal hæmatemesis has been recorded (Bignon), and also perforation with acute peritonitis (Cazin).

Hæmatemesis is very rare in infancy. Apart from its association with melæna neonatorum and the other causes already mentioned, it is seen occasionally in breast-fed infants in perfect health. On examination it is found that the mother's nipples are cracked and bleeding, and it is evident that the infant has sucked the blood from the nipple, and hence this spurious hæmatemesis.

Malignant Growth. — Only a few cases in infancy and in early childhood have been recorded. Cylindrical-celled epithelioma at the pyloric end of the stomach was found in an infant aged fifteen weeks (Cullingworth). Sarcoma of the stomach at the age of $3\frac{1}{2}$ years has been recorded (Finlayson); the writer found secondary sarcoma of the stomach in an infant aged six months. Lymphadenomatous growth in the stomach has been seen at the age of eighteen months (Rolleston and Latham).

Softening of the stomach (gastro-malacia), like the similar but less common condition in the œsophagus, was at one time thought to be an ante-mortem phenomenon, it is now almost certain that it is entirely the result of post-mortem digestion. It appears to be commoner in children than in adults. The softening is generally at the cardiac end of the stomach, which is much thinned, and has a greenish brown colour, while on the inner surface the black arborescent vessels of the mucosa are seen; occasionally a ragged perforation with thinned, almost diffluent edges occurs, and partially digested food is found in the peritoneal cavity.

Disorders of Digestion

The disorders of digestion may perhaps be considered most usefully under the headings of their prominent symptoms. Such a method may seem less scientific than the use of a pathological classification, but for the clinician, at least, it has the advantage of corresponding with what is certain rather than with what is often quite a matter of speculation. In a very large number of the cases of gastro-intestinal disorder in infancy it is impossible to determine whether the disturbance is functional, catarrhal, or even associated with such gross lesions as ulceration or membranous inflammation. While, therefore, it must be recognised that there are such pathological differences, and that in some cases they are recognisable clinically, the subject will be considered here mainly from a clinical standpoint.

Flatulence and Colic.—These two conditions are closely associated and are extremely common in infancy. The accumulation of flatus in the stomach or in the intestine is almost always the result of improper feeding; it may be that one or other constituent — casein, starch, or sugar—is in excess, or the food may be given in too large quantities or at too short intervals. Flatulence may be due to air drawn into the stomach by sucking at an empty bottle, or at a perforated teat used as a "comforter." But sometimes no cause whatever can be found; a child fed at the breast with the utmost care may suffer from flatulence and colic, and that too when the breast milk appears to be good. In such cases one can only suspect that the milk may not be so good as it looks; it may contain too much fat, or its composition may be affected by some disturbance of the mother's health; sometimes it is evidently thin and watery.

Colic sometimes occurs where there is no flatulence. Coldness of the extremities and insufficient covering for the abdomen seem responsible for it in some cases; the presence of undigested food, and especially of masses of constipated fæces in the intestine, causes it in others. Occasionally it may be due to the passage along the ureter of small uric acid concretions, such as are found not very rarely in the pelvis of the kidney in infants.

Symptoms.—Colic in an infant is easily recognised. The child screams during the attack and draws up its legs, the muscles of the abdominal wall being hard and contracted at the time. This may occur at intervals for an hour or more, and then perhaps after the passage of flatus from the mouth or bowel the child becomes easier; or the screaming may continue until from sheer exhaustion the child becomes quiet and falls asleep. Such attacks are very apt to occur in the evening, and the child may be quite free from them during the day. When the colic is severe, other symptoms are often noticed; the infant becomes pale or livid grey about the lips, and there is often slight twitching of the lips, or the eyes are rolled upwards, or the limbs become rigid and the fists clenched. This convulsive tendency may go further, and general convulsions occur. A weakly infant after an attack of colic sometimes becomes quite pale and alarmingly collapsed.

Diagnosis.—It is not always easy to determine the cause of colic in infants. In a large number of the cases some indication of the cause is found in associated symptoms of chronic dyspepsia or gastro-intestinal catarrh, such as chronic vomit-

ing or diarrhœa and marasmus; sometimes the sour smell of the eructations points to fermentation in the stomach; often the relation to meals is obvious, each attack occurring soon after the infant has been fed. It is always wise to examine the abdomen in these cases; the flatulent distension of the stomach or the bowel may be distinctly seen and felt, or scybala may be detected in the intestine. The possibility of intussusception must also be remembered.

Treatment.—In a breast-fed infant the frequency and quantity of the feeds must be regulated, and any defect in the mother's health must be treated. It is hardly ever necessary to wean altogether, but if the milk seem to be poor it may be advisable to substitute hand-feeding two or three times a day, continuing suckling at other times. In hand-fed infants the diet must be carefully revised (*see* "Infant Feeding"). Usually the casein must be diminished or the curd made more digestible by dilution with lime water, oatmeal water, or barley water. Some of these infants are suffering from the results of a diet which contains much starch, especially some of the patent "Foods"; starch, in general, is to be avoided. Where there is evidence of fermentation in the stomach, stomach-washing may be useful; and if the stools are offensive, grey powder with soda is likely to be effectual. The presence of scybala requires the use of an aperient enema and subsequent laxative treatment. A teaspoonful of dill water given just before a feed or mixed with it, or some carminative mixture, such as Tr. card. co. or Spir. ammon. aromat., with bicarbonate of soda and some aromatic water, sometimes prevents the attacks. When the flatulence appears to be chiefly intestinal, creasote (♏ $\frac{1}{4}$-$\frac{1}{2}$) or resorcin (gr. ij.-iv.) may be given three or four times a day. In all cases it is important that the abdomen and thighs should be warmly covered.

During the attack hot flannels should be applied to the abdomen, the feet should be warmed by putting them in hot water if necessary, and the child's position should be shifted frequently to encourage the passage of flatus. This may be assisted by friction or pressure over the abdomen, and the relief afforded in many cases by the prone position as the child lies over the nurse's knee or shoulder, is no doubt due partly to the pressure thus applied. A teaspoonful of hot water with a few drops of brandy or a little aqua chloroformi may give relief, and sometimes a small teaspoonful of salad oil will stop the pain. An enema of soap and water should be given at once if scybala are present, and in any case an enema of plain warm water may do good. If necessary a drop or two of laudanum may be added to the enema, but the use of opiates, particularly in the form of patent "soothing powders" or "syrups," is, as a rule, strongly to be discouraged, although under medical supervision minute doses of Pulv. ipecac. co. (gr. $\frac{1}{4}$-$\frac{1}{2}$ for an infant of four to nine months) are sometimes advisable for a few days, especially where the attacks are followed by much prostration.

Hiccough is frequently troublesome in infants, especially during the first few months of life. It is associated usually with some digestive disturbance, particularly with the condition of flatulence described above. Often, however, no such cause can be found, and there is nothing to account for the onset of this spasmodic action of the diaphragm.

The attacks of hiccough in some cases occur almost daily, and although not serious, are sufficiently distressing both to the infant and to its parents to require treatment. When the attack is prolonged a weakly infant is sometimes quite exhausted by it, and even in milder degrees the discomfort it causes to the child is often shown by a fretful cry as each spasm occurs.

Treatment.—When the attacks are frequent the diet should be carefully considered; it may be that some slight alteration, such as has been recommended in the cases of flatulence, may prevent the hiccough. The attack may be shortened sometimes by very simple methods such as changing the baby's position at short intervals, gentle patting on the back, or friction over the upper part of the abdomen. In other cases a teaspoonful of hot water, either alone or with one or two drops of sal volatile, or five or ten drops of brandy, may be successful. The administration of a weak acid, such as lemon juice, or one or two drops of dilute sulphuric acid in a teaspoonful of cold water, is sometimes very effectual.

Vomiting.—Under this head must be mentioned first the regurgitation of food, which is so common during the first two or three months of infancy as to be almost physiological; it might perhaps be distinguished as *repletion-vomiting.* This is seen in infants who take their food greedily whether at the breast or from the bottle. A few minutes after, sometimes even before the meal is finished, they regurgitate without effort and without discomfort a small part of the contents of the stomach. This vomiting seems to be really a protective arrangement to prevent over distension of the small infantile stomach. As a rule it has no ill effect, and the child gains weight steadily in spite of the vomiting. Nevertheless it is wise to check this regurgitation by reducing the quantity of each meal, for the continual over-feeding is apt to lead to actual gastric catarrh, and sometimes certainly, as Dr. Goodhart suggests, it seems as if a habit of vomiting were established which it is difficult to break.

Apart from such repletion-vomiting, the

commonest cause of vomiting in infancy is some form of gastric irritation. In many cases the feeding is faulty, either as to quantity, quality, or frequency of food. Sometimes the irritation appears to be toxic, as where vomiting is due to sour milk or bad eggs, and it is probable that the vomiting which occurs in severe cases of summer diarrhœa is sometimes, at least, toxic in origin. Vomiting is very frequently associated with diarrhœa, indeed it is comparatively seldom that an acute attack of vomiting occurs in an infant without some intestinal disturbance. Vomiting in infancy is rarely obstructive; the possibility of some congenital narrowing of the pylorus or duodenum, however, must be borne in mind in any case of persistent vomiting in early infancy (*vide* Congenital Hypertrophy of the Pylorus): intussusception also, strangulated hernia, and the rarer forms of acute intestinal obstruction must not be forgotten.

Apart from disorders of the gastro-intestinal tract, vomiting is a common symptom of the onset of many acute diseases in infancy; it may also be due to some intracranial disease, meningitis, or tumour.

An *acute* attack of vomiting in an infant is perhaps most often the result of acute dyspepsia. The infant seems ailing; it lies quiet and pale, or cries with colicky pains. The temperature may be raised to 101° or 102°. Vomiting occurs at short intervals, and lasts a few hours, ceasing when the stomach is emptied. There is usually some slight transitory diarrhœa. This begins soon after the vomiting has commenced, and often lasts a day or two after the vomiting has ceased. This may constitute the whole illness in a mild attack, but in more severe cases, or where the irritation is increased by the continuance of improper food, the vomiting may continue even after the food has been rejected, and mucus, sometimes bile-stained or even tinged with blood, may be vomited. Diarrhœa also in such cases is more severe, and the constitutional disturbance is considerable. The infant quickly becomes hollow-eyed with sunken fontanelle, and lies apathetic and exhausted.

In these severer cases there is probably usually some inflammatory or catarrhal change in the mucous membrane of the stomach, and the term *acute gastric catarrh*, or even *gastritis*, might properly be applied to them.

Prognosis.—In young infants such an attack is always serious and may end fatally; but in most cases, if the cause is removed, the illness ends favourably. Infants who have had such an attack are very liable to relapse with the slightest error in feeding; and sometimes recovery is even less complete. The vomiting becomes less frequent, but does not disappear, the attack ceases to be acute, but becomes still more troublesome as a chronic condition.

Chronic vomiting is common, especially in hand-fed infants. In many cases, no doubt, it signifies a *chronic gastric catarrh*, but in many more it is due to a chronic dyspepsia, a functional disturbance, without organic changes, in the mucous membrane. Vomiting may persist for many weeks with no evidence whatever of organic lesion, and all one can say is that there is undue irritability of the stomach. As mentioned above, it seems likely that a morbid habit may be established which may account for the intractability of some cases.

To whatever cause the vomiting be due, except in the almost physiological repletion-vomiting of early infancy, the child soon begins to lose weight, its growth is arrested, it becomes pale and fretful. The appetite is sometimes large, sometimes diminished, and often there is pain and screaming after meals. The tongue may be furred, and sometimes the breath has a sour odour, and there are sour-smelling eructations. The bowels are often irregular, attacks of diarrhœa alternate with costiveness; but in many cases there is a chronic diarrhœa, and the condition is really a chronic gastro-intestinal catarrh (*vide* Chronic Diarrhœa, p. 412). Vomiting may occur two or three times a day, or after almost every meal. Unlike the repletion-vomiting which occurs immediately after, or even before the meal is finished, the vomiting of dyspepsia or catarrh usually follows after an interval of a quarter or half an hour, or more. In the worst cases the infant gradually becomes weaker and more emaciated; the skin is dry and hangs in folds; thrush appears in the mouth, and either from exhaustion or from broncho-pneumonia, or from some other complication, the child dies. In other cases the child slowly recovers, but remains delicate, and requires the utmost care in feeding for many months.

Morbid Anatomy.—As a rule the stomach shows nothing abnormal to the naked eye. It may be in a state of dilatation or contraction. In the worst cases, those of acute gastritis, the mucous membrane sometimes shows intense purplish congestion, usually over a limited area, sometimes also small hæmorrhages are seen in its substance, and very rarely small follicular erosions, while shreddy brownish material, probably altered blood or blood-stained mucus, is found adhering to it. The microscope shows shedding of superficial epithelium, small-cell infiltration both of the superficial and of the deeper parts, and considerable dilatation of vessels. In the more chronic cases, where no change whatever is detected by the naked eye, microscopic examination often reveals definite changes in the mucous membrane, especially when the viscid, mucus-like character of the vomit during life has suggested some gastric catarrh. The changes are similar to those found in the acute cases, except that they are

less in degree, and the inflammatory infiltration shows more or less advanced organisation with the development of fibrous tissue which has partially replaced the glands of the mucosa. They are, in fact, exactly similar to those found in the intestine in some cases of diarrhœa (*vide* p. 415). The importance of these changes is in the light they throw on the intractable wasting which too often follows chronic vomiting in an infant.

Dilatation of the Stomach is present in some of these cases; perhaps especially where a chronic gastric catarrh is associated with rickets; the writer has found considerable dilatation in such cases. It has been shown that in the chronic dyspepsia of infants the food remains in the stomach longer than normal, and sometimes even clinically enlargement of the stomach can be detected, but anything like a chronic dilatation of the stomach, such as would give rise to the characteristic symptoms associated with such a condition in adults, is practically unknown in infancy, except in such obstructive conditions as congenital hypertrophy of the pylorus.

Treatment.—Vomiting, gastric in origin, is nearly always due to some fault in feeding, and sometimes all that is needed is a careful revision of diet. Where it is a symptom of diseases other than those of the gastro-intestinal tract, usually no special treatment is required. In an acute attack of vomiting due to faulty feeding, the evacuation of the irritant is usually sufficiently accomplished by the vomiting, but in some cases recovery may be hastened by making the evacuation more thorough, either by washing out the stomach through a soft catheter passed through the nose or mouth, or, as recommended by Dr. Holt, by giving the infant warm water in large quantity as an emetic. In severe cases of vomiting it is advisable to stop all feeding by the mouth for a few hours, and then to feed only with very minute quantities at short intervals. A teaspoonful of cold water with 5-10 drops of brandy every half-hour will be quite enough for a few hours; sometimes hot water seems more useful. Infants at the breast must only be allowed to suck for a very short time, and in some cases it is better to try albumin water or whey in small quantities for some hours before returning to the breast. Albumin water (the white of one raw egg cut up with scissors and shaken with half a pint of water, strained, and flavoured with a small quantity of cinnamon water or dill water) is often useful for a short time in any case of acute vomiting. Few foods are more valuable either in acute or chronic vomiting than whey, especially sherry-whey (made by heating half a pint of milk just to the boiling-point, then adding a good wine-glassful of sherry, heating to boiling-point again, and when the curd has settled, straining through muslin). A teaspoonful, gradually increased to a tablespoonful, of whey may be given frequently, and sometimes the addition of one or two drops of sal volatile to the plain whey checks the vomiting. When the attack has passed off the digestion is often much impaired, casein particularly seems to be digested with difficulty, and it may be necessary to peptonise the milk, or to use a humanised milk for a time.

In chronic vomiting the feeding is generally the most important consideration. Sometimes one food, sometimes another is successful, and it is often necessary to try several before a suitable one can be found. Whey, peptonised milk, humanised milk, prepared foods, especially the malted foods, are all useful at times. Whatever food is given, it must be given in small quantities, at short intervals, according to the age.

In any case of vomiting in which all food is persistently rejected, feeding through a tube passed down the œsophagus, either from the nose or mouth, is worthy of trial.

Sedative drugs are often of value, especially in acute cases after the stomach has been emptied. A mixture of bismuth and soda is generally useful, and a small dose of tinct. opii (♏ $\frac{1}{4}$-$\frac{1}{2}$ three times a day for an infant six months old) may be added to this, or hydrocyanic acid (♏ $\frac{1}{4}$ for an infant four months old) with sod. bicarb. and some aromatic may be given.

In the chronic cases these drugs are of less value; and small doses of grey powder given two or three times a day, alone or with soda, are more likely to be useful; sometimes papain (gr. j.-ij.) or pepsin (gr. j. with three or four meals a day) will do good by assisting digestion. Where the vomit is particularly sour or frothy, creasote is sometimes very effectual. Very severe and persistent vomiting, especially in acute cases, may sometimes be checked by counter-irritation in the shape of a mustard plaster to the epigastrium.

The value of stomach-washing is perhaps hardly sufficiently appreciated. Vomiting may be checked by only one or two washings with plain hot water or with a weak solution of sod. bicarb. (gr. ij. to ℥j.). This is specially useful where the presence of mucus in the vomit shows that there is some gastric catarrh; where there is evidence of fermentation, resorcin (1 in 1000) may be used for the washing.

Recurrent Vomiting (*Periodic or Cyclic Vomiting*).—It is convenient to mention this rare disorder here, although it is probably rather a nervous than a digestive disorder. It begins sometimes in infancy (in one case under the writer's notice it began at the age of six months), but more often after the first dentition. It is most common in children of nervous, excitable temperament.

Symptoms.—At intervals of a few months attacks of vomiting occur without apparent cause. The child is languid and depressed.

Its face is sallow, the bowels are costive, and in some cases the motions are pale as if there were some deficiency of bile. Every attempt to take food is followed almost immediately by vomiting; retching is sometimes violent. The temperature may be normal throughout or somewhat raised, especially at the onset. After two or three days the vomiting usually ceases, and the child rapidly recovers, but sometimes the attacks last ten days or longer, and exhaustion may be so great as to threaten life. Such attacks may recur for several years; in the case mentioned above the attacks had recurred three or four times a year for five years.

It seems probable that these attacks are nervous in origin; certainly they seem to be little if at all influenced by such treatment as is useful in the vomiting of gastric catarrh. It has been suggested that they resemble migraine in their pathology, and Dr. Gee records a case in which the mother suffered from typical migraine.

Diagnosis.—The nature of a first attack can scarcely be recognised; the absence of any apparent cause and the intractability of the vomiting might suggest that it was of this character. A diagnosis can only be made with certainty after repeated attacks have shown the tendency to recurrence. To distinguish it from early meningitis may be extremely difficult, but headache is usually absent in recurrent vomiting, and is never likely to be so severe as in meningitis; later the absence of paralytic symptoms and of ophthalmoscopic changes will assist the diagnosis.

Treatment.—In the intervals all excitement and over-exertion, bodily and mental, especially school pressure, must be avoided. Healthy outdoor exercise short of fatigue should be encouraged. Diet should be simple. Intermittent courses of arsenic and bromides should be given.

During an attack the strength must be supported by careful feeding and stimulants if necessary. Fluids in very small quantities, iced or peptonised, may be tried, but in severe cases rectal feeding is necessary. The bowels should be opened freely by repeated small doses of calomel. Sedatives such as bromides, belladonna, or hydrocyanic acid, or counter-irritation to the epigastrium, may be tried, but usually the vomiting runs its course uninfluenced by drugs.

Diarrhœa

(*Gastro-intestinal catarrh; Gastroenteritis*)

Under this head will be described those disorders of the gastro-intestinal tract in infancy in which diarrhœa is the prominent symptom. In a large proportion of cases, however, the stomach is affected with the intestine, and vomiting may be quite as prominent a symptom as the diarrhœa. Various classifications of these disorders have been attempted, some based on clinical, some on pathological grounds, but the subject is one of great difficulty, for while it is evident that the cases fall into certain clinical groups, these groups are by no means well defined, and pathologically they tend to run one into the other. Diarrhœa may be acute or chronic. Acute diarrhœa may perhaps most usefully be divided into (1) Simple, (2) Inflammatory, (3) Choleraic.

Ætiology.—Before describing the various forms of diarrhœa it may be well to say something about their general ætiology; for our knowledge on this point is at present so vague that it would be useless to attempt to associate particular causes with particular forms except in a very general way.

There is no age at which diarrhœa is more prevalent or more fatal than that of infancy; but the liability is much greater in the first year of life than in the second, the mortality also is much heavier in the first year. Statistics taken by the writer at the Hospital for Sick Children showed that 90 per cent of the fatal cases of infantile diarrhœa were under twelve months old. Girls and boys are about equally affected.

The influence of season is very marked. Scattered cases occur throughout the year, but during the hot summer months there is an enormous increase, both in the prevalence and in the mortality of infantile diarrhœa. The actual seasonal variation in any particular place or year varies with the meteorological conditions; in London the maximum prevalence would seem to be usually from the beginning of July to the middle of October.

There is manifestly a very close relation between a high mean temperature of the atmosphere and infantile diarrhœa, but what this relation may be it is difficult to ascertain exactly. Probably the high atmospheric temperature favours the growth of certain bacteria which are capable of exciting diarrhœa; these reach the intestine in the food, particularly in cow's milk which has not been properly prepared. But what the source of these bacteria may be is unknown; it has been suggested that they are from the soil, and it has been noticed that the summer increase of diarrhœal mortality follows the curve of soil-temperature as shown by the four-foot earth-thermometer (Ballard).

At any time of the year infants who are entirely or even partially hand-fed are much more liable to diarrhœa than those who are entirely breast-fed; and this difference is no doubt due chiefly to the almost complete sterility of human milk as drawn by the infant from the breast, while fresh cow's milk as supplied commercially almost invariably contains some bacteria.

But other factors are always present in the diarrhœa of hand-fed infants; the food is often,

especially amongst the poorer and more ignorant classes, of a most indigestible kind; and in all classes it is apt to be given in too large quantity and at too short intervals.

The mortality from infantile diarrhœa is greater in town than in country districts, but it would seem that this difference does not depend entirely on density of population; poverty and defective sanitation are potent causes.

There can be little doubt that infantile diarrhœa is sometimes contagious; cases have been observed where it has seemed to spread from infant to infant, and also to nurses, but the possibility of a common source of infection, especially in the milk-supply, must be carefully excluded.

Of recent years attempts have been made to isolate from the very numerous bacteria which are always present in the intestine a specific micro-organism as the cause of the diarrhœa, especially the summer diarrhœa of infants, but hitherto the results have been quite inconclusive.

Dr. Klein has recently isolated a micro-organism, the bacillus enteritidis sporogenes, which there is reason to believe plays an important part in the causation of some of the acute cases of summer diarrhœa; but other micro-organisms have also been found with more or less constancy, and it seems probable that there may be several different micro-organisms which are all equally capable of exciting a gastro-intestinal catarrh.

The manner in which these bacteria cause diarrhœa is also probably not the same in all cases: in some they produce chemical changes in the food either outside the body or in the intestine, so that the altered food is an irritant; in others they themselves produce toxins which not only act as an irritant to the mucous membrane, but also when absorbed may cause some of those profound disturbances, especially of the nervous system, which are so marked in some cases of diarrhœa.

But while it is almost certain that a very large proportion of the cases of diarrhœa in infancy are due to micro-organisms, and are therefore "infective,"—and it must be understood that the mildest summer diarrhœa and the most virulent choleraic diarrhœa are in this sense equally "infective," though probably due to different micro-organisms,—one recognises clinically many cases in which there is no reason to suppose any such cause at work.

Indigestible food in the intestine may act as a mechanical irritant. Certain chemical poisons are sometimes the cause of diarrhœa, and apart from the ordinary aperient medicines some drugs given medicinally for other purposes, *e.g.* ammonium carbonate or arsenic, may act thus.

Most of the specific fevers are occasionally associated with diarrhœa; thus whooping-cough, measles, scarlet fever, and diphtheria are all sometimes complicated by severe diarrhœa. Typhoid and tuberculous ulceration of the intestine will not be considered here, as they fall into a somewhat different category, but in any given case of diarrhœa they must, of course, be considered as possible causes.

Diarrhœa is very common with broncho-pneumonia in infancy; and it is often difficult to be sure which was the primary disease; here, as in the specific diseases just mentioned, the diarrhœa may be "infective" in character, and perhaps the same explanation applies to the diarrhœa which often complicates suppurative conditions, such as pyæmia or empyema.

The part played by dentition in the causation of diarrhœa is obscure, but it is certain that the eruption of a fresh tooth is frequently associated with diarrhœa; possibly the nervous disturbance which so often accompanies dentition may, like fright or excitement in older children, affect the intestine.

Lastly, the effect of chill must be mentioned; chronic diarrhœa especially sometimes seems to be prolonged, if not actually caused, by exposure to cold, and in infancy this cause is too often present owing to the insufficient covering for arms, legs, and abdomen which custom demands.

Simple Diarrhœa (*Intestinal Indigestion, Gastro-intestinal Catarrh*).—In its mildest form simple diarrhœa is quite a transient and slight affection. The infant, who has previously been quite well, seems restless, and perhaps shows signs of colicky pain, screaming and drawing up its legs. The bowels are open five or six times in the day; the fæces at first are only slightly looser than normal, and probably contain undigested casein or other food-stuff, but soon they become more liquid, and perhaps green and offensive with some mucus. Frequently some vomiting precedes the diarrhœa, showing that there is some gastric as well as intestinal irritation. The temperature may be raised to 100° or 101° at first, but quickly becomes normal, and often is normal throughout. Even with a very mild attack the infant quickly shows that it is ill; it ceases to be playful, and if it has already begun to crawl or walk, it may give up these accomplishments altogether; as the mother says, "it has gone off its legs," it only wants to lie quietly in its cot or in its mother's arms. At the same time it is pale and dark under the eyes; its tongue is slightly furred, and it may refuse its food, or drink greedily as if it were thirsty.

But the condition rapidly improves; sometimes after only a few hours, sometimes after two or three days, the stools become less watery, and gradually resume their normal colour and consistency. The infant remains pale and fretful for a day or two after the diarrhœa has ceased, but speedily regains its usual health.

Such is a mild attack of diarrhœa in an

infant; and where the illness is due, as commonly happens, to some irritating food-substance in the intestine, the evacuation of the irritant usually brings the attack to an end. Too often, however, these mild symptoms do not pass off as soon as one expects, the diarrhœa continues day after day, and what began as a simple diarrhœa becomes the more serious condition which is called inflammatory diarrhœa.

Inflammatory Diarrhœa (*Febrile Diarrhœa, Gastroenteritis, Ileocolitis*). — Such a heading necessarily includes several conditions which are more or less distinct pathologically, but as these cannot with any accuracy be distinguished clinically, it seems more useful to describe them together. Many cases of "summer diarrhœa" are of this variety.

Often the condition begins like a simple diarrhœa, but instead of subsiding in a few days the diarrhœa continues; the stools are liquid, greenish, or dark brown and offensive, and often show some mucus and streaks of blood. The bowels are open many times a day, and the buttocks and perinæum are soon red and raw with the irritation of the fæces. It is remarkable how quickly nutrition suffers in these cases; after even one day of diarrhœa, if it be at all severe, the muscles feel less firm, and the face looks less full, and in two or three days there is distinct loss of flesh, the child is pale and hollow-eyed, and the fontanelle depressed. The tongue is often quite clean, in fact almost abnormally clean and red, or slightly furred with very red papillæ projecting through the white fur.

The temperature may be raised at the beginning of the illness, but afterwards in the less severe cases it is normal; if the diarrhœa be prolonged, and especially if there be much vomiting, it may be subnormal.

After two or three weeks of such symptoms the diarrhœa may gradually cease, and the child slowly mend; but at any time in such an attack, and sometimes, indeed, from the beginning, more severe symptoms may occur. The temperature rises and there is irregular pyrexia; the infant looks acutely ill, it is drowsy and apathetic, the cheeks are flushed, the tongue is furred. The abdomen is often full, and sometimes distinctly tender on palpation, especially along the course of the colon. The stools are very frequent, perhaps ten or twelve a day, and mucus stained a reddish brown or streaked with bright red blood is commonly present, pointing to affection especially of the colon. Vomiting is frequently associated with the diarrhœa, even where the lower part of the intestine seems to be chiefly affected.

Dr. Eustace Smith attaches considerable importance to loss of elasticity in the skin in these cases as evidence of deficient renal excretion, and undoubtedly the urine is diminished, as might be expected where food is so rapidly hurried through the intestine and so little absorption takes place. It is, however, equally certain that any actual nephritis is an exceedingly rare complication, in spite of the fact that a small amount of albumin in the urine is very common in all forms of diarrhœa in infancy.

Œdema not infrequently occurs, especially in the extremities, after the diarrhœa has lasted a week or more; this again must not be taken as necessarily indicating any renal complication; in some cases where it is marked the urine is perfectly normal, and even if albumin be present it is usually very slight; it seems probable that the œdema is rather due to a flagging circulation, combined with some hydræmic condition of the blood, and possibly with some degenerative change in the vessels.

Eventually exhaustion becomes extreme; the infant lies quite apathetic, almost in a stupor, with sunken eyes and depressed fontanelle; the eyelids are only half-closed in sleep, and a film of muco-pus collects on the cornea, which may become dry and ulcerated. The extremities are cold, the pulse is feeble and very rapid. The temperature, as the infant lies in this state of collapse, may be subnormal, but often when taken in the rectum it is found to be high; there may even be hyperpyrexia in spite of the coldness of surface. Death results usually from exhaustion, but in some cases is preceded by convulsions, or by symptoms like those of meningitis.

These cases, however, are by no means always fatal; the diarrhœa may gradually cease and pyrexia disappear, and the infant may gradually regain its former nutrition. Too often, however, recovery is less complete; sometimes, although the acute diarrhœa has ceased, the bowels continue to be open three or four times a day, or are irregular, constipation alternating with looseness; the fæces show undigested curd, and are perhaps greenish and offensive with some mucus; the child gradually becomes puny and wizen-faced, and after weeks or months of slow emaciation dies of exhaustion. Sometimes, although the bowels become regular, the infant steadily wastes, and no manner of feeding seems to influence the wasting which eventually proves fatal. In other cases, after the acute diarrhœa has been checked, the child seems to have made a good recovery, but for months, and sometimes even for years afterwards, the slightest fault in diet causes some diarrhœa, or the appearance of mucus in the stools with more or less disturbance of general health.

Choleraic Diarrhœa (*Cholera Infantum, Septic Diarrhœa*). — This form of diarrhœa is more especially limited to the summer season than are the preceding forms. The name is usually given to any case of summer diarrhœa in which the stools are watery, and the illness runs a very acute course, lasting only a few days or even a few hours. It has been sug-

gested that the name should be reserved for those cases in which the stools have a rice-water appearance, but as this symptom does not, so far as our present knowledge goes, indicate any special bacteriological difference, and may or may not be present in cases which otherwise run an exactly similar course, there seems to be no reason for adopting this limitation. It will, of course, be understood that there is no connection between so-called "cholera infantum" and Asiatic cholera.

Symptoms.—These, as already mentioned, are characterised by their extreme severity and acuteness. In the worst cases, an infant, often well nourished and apparently in perfect health, is suddenly seized with diarrhœa. The stools, at first semi-liquid and yellowish, rapidly become thin and watery, sometimes brownish and very offensive, usually almost colourless, like rice-water, with very little smell. Vomiting is severe, all food is rejected almost directly it reaches the stomach. In a few hours the alteration in the face is striking, the plump pink cheeks have become pale and flabby, the eyes are deeply sunken, and the brows often knitted as if with pain. The fontanelle is deeply depressed, the tongue is dry and brownish red, and the infant is continually licking its lips in its distressful thirst. The abdomen is deeply hollowed out, and the skin over it lies in loose, wrinkled folds. The child flings its arms restlessly about, and rolls its head from side to side, or lies in an apathetic condition, taking no notice of its surroundings, and gradually sinking into actual stupor. Respiration is often sighing; the temperature is usually high, but may be subnormal. The urine is diminished, and may even be suppressed for many hours, and often shows more or less albumin.

After two or three days, or, in the worst cases, after only a few hours, exhaustion is extreme, the extremities become cold and blue, the face, and especially the nose, has a pinched look and grey colour, the pulse becomes very rapid, 180-200, and barely perceptible at the wrist, and the child dies.

The course of the disease varies somewhat in different cases. The onset may be less acute than that described above; the infant is ailing for a few days with what appears to be a simple diarrhœa, and no alarm is felt until a sudden increase in the severity of the diarrhœa, with the appearance of watery colourless stools and the onset of uncontrollable vomiting, shows how serious the illness is. In other cases the choleraic symptoms supervene in the course of a chronic diarrhœa or intestinal indigestion which has lasted for many weeks or months, the temperature which has hitherto been normal suddenly rises, diarrhœa and vomiting are profuse, and death occurs in a few days. (*See also* "Cholera Nostras," vol. ii. p. 125.)

CHRONIC DIARRHŒA is in many cases the sequel of acute diarrhœa, but it may occur independently of any acute attack, and may be quite insidious in its onset. In these insidious cases the diarrhœa is perhaps most often the result of faulty feeding; it is, in fact, the manifestation of a chronic dyspepsia. In some there is also some constitutional defect present, sometimes syphilis, but much more often rickets, which is so powerful a predisposing cause of catarrh of mucous membranes. Chronic diarrhœa is not uncommon as a sequela of whooping-cough and of measles. In the children of the poor, exposure to cold and generally faulty hygiene play an important part in its production. Under this head come many of the cases of so-called "consumptive bowels," a vague, inaccurate term which it is to be hoped will die out ere long; very few of the cases to which it is applied show any tuberculous lesion, and many of those who use the term simply mean by it a disorder of the bowels associated with wasting. Chronic diarrhœa may be the result of tuberculous ulceration of the intestine, but this is quite the exception in infancy.

Symptoms.—The diarrhœa may have persisted since an acute attack, probably as a much slighter looseness of the bowels, which are open four or five times a day, with some undigested food in the fæces. But often no definite date can be assigned for its onset, and so insidious may it be, that the mother has only noticed the wasting, the condition of the bowels has not attracted her attention. On inquiry, it is found that the bowels are open too frequently, and the stools are unhealthy. The character of the stools varies considerably. In some cases of chronic diarrhœa the stools are semi-liquid, deep brown, and very offensive; in others they are pale yellowish or drab coloured, and pultaceous; in others again there are small lumps of white curd mixed with very small pieces of dark green fæcal material very like chopped spinach. The green colour is probably due to the presence of biliverdin; alkaline decomposition in the intestine changes bilirubin to biliverdin (Pfeiffer); the fæces may be acid when passed, but the acidity is not sufficient to change the colour. The suggestion that a specific micro-organism causes the colour (Lesage) lacks evidence.

The presence of mucus, especially if stained with blood, points to affection of the colon, and if the blood occur in bright red streaks, it is probable that the lower portion is specially affected. Those cases in which the colon is much involved are further distinguished by the presence of tenesmus, sometimes by some tenderness over the large intestine, and also by a tendency to prolapse of the rectum, which is, however, more marked in children two to four years of age than in actual infancy.

In all cases the general health suffers considerably. The infant, who was good-tempered and playful before, becomes fretful, miserable,

and apathetic. If he be at the age of learning to talk, he ceases to make progress, and may lose entirely the few words he has acquired, and a child who has just learnt to walk with the aid of a chair may even be unable to stand. Nutrition is impaired very quickly. The infant rapidly wastes, the skin is dry and inelastic, and can be pinched up in loose folds. After a few weeks of chronic diarrhœa the emaciation is often extreme, the face is shrunken, and looks aged and wrinkled, there is a deep furrow passing downwards from just above the ala nasi on each side, and forming a crescent round the angle of the mouth, so that the lips and mouth seem to be prominent, and the general appearance is not unlike that of a monkey. The skin, both of the face and trunk, is sometimes of a muddy brownish colour, sometimes very white, the edges of the eyelids look red and sore, and there is frequently some eczema behind the ears. The fontanelle is depressed, the cry is a feeble whine, the extremities are often cold and blue. The tongue is quite clean in many cases, but may be slightly furred. The abdomen is usually retracted, and through its wasted wall the coils of intestine can be seen; slight peristalsis may also be visible. Sometimes the abdomen is full from the presence of gas in the intestine, and at the end of the illness an acute abdominal distension may occur and hasten the fatal result. Flatulent eructations are often present, and sometimes have an extremely disagreeable sour odour. As in other forms of diarrhœa, especially when there is any neglect in changing the diapers and in keeping the parts clean and dry, the buttocks and perineum are apt to become red and excoriated.

In this condition, perhaps, more commonly than in acute diarrhœa, œdema first of the extremities and then of the whole body is likely to occur, and is usually a symptom of the gravest significance; purpura also in spots or larger patches is very common towards the end of the illness, beginning often on the abdomen, or on the upper part of the chest or neck. After these symptoms have appeared the end is not usually long delayed.

Sooner or later the diarrhœa becomes more severe, the bowels are open ten or eleven times a day, and the evacuations are more liquid; vomiting, which in many cases has only been occasional and slight, or may have been absent throughout the illness, now becomes frequent, and the temperature which has hitherto been normal may rise, or may drop to subnormal. In some cases there is irregular pyrexia for several days before death; in others the temperature remains normal until a few hours before the fatal issue, when it rises to 107° or 108°. With the onset of these symptoms the child falls into the state of collapse which has already been described, and death results as a rule from exhaustion.

But although chronic diarrhœa in an infant is always a serious condition, and always calls for a very guarded prognosis, it is not always fatal. With careful treatment the bowel may be coaxed back to a healthier action, and the child slowly recover, but in such cases, even more than in those which recover from inflammatory diarrhœa, the child is likely to be delicate for a long time after the attack, and relapses are extremely common.

Complications.—In many cases of diarrhœa, whether acute or chronic, but especially in the more severe cases of acute inflammatory diarrhœa, bronchitis and broncho-pneumonia are very liable to occur. The association is always a dangerous one, and in some cases the fatal result appears to be directly due to the broncho-pneumonia. In chronic diarrhœa the infant is often so feeble and the broncho-pneumonia comes on so insidiously, that it produces but little alteration in the symptoms, and may only be discovered post-mortem.

Collapse of the lung, although very commonly found after death in fatal cases of diarrhœa, can seldom be detected clinically.

Thrush is frequently associated with diarrhœa, especially in the more chronic cases; it is of less serious import than mothers usually imagine, but where the soreness of the mouth interferes with the taking of nourishment it may add to the seriousness of the condition.

Otitis media is liable to occur with diarrhœa as with any other severe illness in childhood, no doubt owing to the prostration favouring the accumulation of micro-organisms in the pharynx and their extension up the Eustachian tubes. The complication is worth bearing in mind, as it may simulate cerebral symptoms and cause much distress, which is easily relieved by hot applications, or, if necessary, by puncture of the membrana tympani. Nervous complications are not rare in severe diarrhœa. The commonest are convulsions. These sometimes occur at the onset of the disease, but much more often during the last few days of life. In many cases the child falls into a sort of continuous convulsive state, rather than an actual convulsion; it lies apparently unconscious, with its eyes turned upwards or to one side, its hands and legs twitching slightly, and an occasional twitch of the facial muscles, and in this condition it may remain for some hours before death occurs. The occurrence of convulsions in the course of a severe diarrhœa is always a very serious symptom.

Sometimes, both in severe cases of inflammatory diarrhœa and in choleraic diarrhœa, where collapse and exhaustion are extreme, the child falls into a condition which resembles in some respects meningitis. It has been called "spurious hydrocephalus." The child becomes almost completely unconscious, the head may be retracted and the limbs rigid, there may be

some strabismus, and even the sudden scream of meningitis may occur, the respiration is irregular, the pulse feeble and rapid; but with all these symptoms the fontanelle is depressed. As a rule such cases are speedily fatal. No naked-eye lesion is found in the brain to account for the symptoms, which it seems probable are due to some toxæmia from the intestinal infection.

Thrombosis of cerebral sinuses is a very rare complication. It occurs usually in cases where there is great exhaustion; it is but seldom recognised during life, the symptoms are very variable; paralysis of cranial nerves, weakness of limbs, and convulsions have been observed, but sometimes there are no symptoms at all.

An exceedingly rare complication of diarrhœa is nephritis. It occasionally occurs in very severe cases of cholera infantum; otherwise, except for some slight cloudy swelling in cases where there has been much pyrexia, the kidney is almost invariably healthy, although, as already pointed out, slight albuminuria is common. It is possible that in some cases this albuminuria is the result of mechanical irritation of the pelvis of the kidney by uric acid sand, which is very frequently found in the urine during life and in the pelvis of the kidney after death in these cases.

Prognosis.—In all cases of diarrhœa in infancy the prognosis must be guarded. The case which at its onset looks like a mild case of simple diarrhœa may in a few days merge into the severest form of inflammatory diarrhœa. At the same time it must be remembered that a simple diarrhœa may be very severe in its onset, and may produce considerable exhaustion, with sunken eyes and depressed fontanelle, especially in very young infants, but after a few hours the symptoms may gradually subside, and in a few days there may be complete recovery. In all forms of diarrhœa, the younger the infant the less favourable is the prognosis. Continued high temperature, whether regular or irregular, in any case of diarrhœa is a serious symptom; it usually means that the case is one of inflammatory diarrhœa, and sometimes points to the grosser lesions, such as ulceration and membranous inflammation, which are found in some cases of inflammatory diarrhœa. Tenderness of the abdomen also points to an inflammatory condition, and when it is well marked is an unfavourable symptom.

Profound nervous disturbance, especially apathy or stupor, is a very grave symptom. Convulsions are serious, but do not necessarily mean a fatal result; their significance is less grave when they occur at the onset of the diarrhœa than when they occur in the stage of exhaustion and prostration.

Choleraic diarrhœa is always a grave disease; a very large proportion of such cases are fatal.

Diagnosis.—In some cases of chronic diarrhœa it is extremely difficult to determine whether the diarrhœa is due to tuberculous ulceration of the bowel. A gastro-intestinal catarrh from faulty diet is very much commoner in infancy than tuberculous disease of the intestine. In infants under six months of age tuberculosis is decidedly rare, and even up to the age of nine months it is not common. The association of irregular pyrexia with a chronic diarrhœa suggests a tuberculous lesion, especially where there is a strong family history of tuberculosis, but the strongest evidence is palpable enlargement of the mesenteric glands or the presence of some definite tuberculous lesion elsewhere. In most cases the question cannot be decided during life, and one can only say that the vast majority of the cases of chronic diarrhœa in infancy are non-tuberculous. Typhoid fever is so rare in infancy, and when it does occur is so much more often associated with constipation than with diarrhœa, that it is hardly likely to cause any trouble in diagnosis; the continuous or remittent pyrexia lasting ten days or a fortnight will usually serve to distinguish it from chronic gastro-intestinal catarrh, and other symptoms of typhoid, enlarged spleen, rose spots, or a positive result with Widal's reaction, may assist the diagnosis.

The frequency with which mucus and streaks of blood are present in the stools in diarrhœa with affection specially of the colon is noteworthy; these appearances do not always indicate an intussusception, but on the other hand the occurrence of an intussusception has been mistaken for the onset of a gastro-intestinal catarrh, and the presence of mucus and blood on the stools has been made light of, with disastrous result. Such an error can be avoided only by careful examination of the abdomen for the presence of the characteristic tumour.

The presence of ulceration in the bowel cannot be determined clinically; it is not even possible to determine with any accuracy during life which part of the intestine is affected. Such terms as ileocolitis, ulcerative or membranous colitis, have a definite meaning for the pathologist, but are of very little practical value for the clinician. In a general way it may be said that an acute course with a very high temperature, the passage of mucus and streaks of blood in the fæces, and tenderness along the course of the colon, point to some intense inflammation of the large intestine, but a case may show all these symptoms during life, and yet show nothing post-mortem beyond slight congestion of the mucous membrane of the intestine.

Morbid Anatomy.—The most striking feature in the morbid anatomy of the diarrhœa of infancy is the extreme disproportion between the naked-eye lesions found post-mortem and the severity of the clinical symptoms. In the most acute form of all, choleraic diarrhœa, the intestine usually shows nothing abnormal beyond pallor of the mucous membrane. In many

cases of inflammatory diarrhœa there is no naked-eye lesion whatever; in many there is only slight congestion of the mucous membrane in parts of the ileum and colon. A few cases show some undue prominence of the solitary follicles in the large intestine, so that its mucous membrane appears to be studded thickly with little round bosses, and in such cases there is sometimes also swelling of the Peyer's patches in the lower part of the ileum. Ulceration is but rarely present (the writer found it in 5 per cent). In some cases small round ulcers are present in the ileum; they are mostly shallow, with no thickening of their edges, and are usually quite independent of the Peyer's patches; sometimes only four or five are present, sometimes a score or more. In other cases a much more extensive irregular ulceration occurs in the colon, and more rarely in the lower part of the ileum; the ulcers are usually superficial, with no thickening at their edges, but the surrounding mucous membrane may be intensely congested and swollen. Such ulceration is often limited to one part of the colon, it may be at the cæcum and on the lips of the ileo-cæcal valve, or in the lower part of the colon, or even in the rectum. A still more rare condition is membranous inflammation of the colon, in which irregular areas of superficial ulceration are covered by a greyish white membrane, very like that seen on the fauces in diphtheria.

In some of the more chronic cases of diarrhœa a curious appearance like that of the chin after shaving is seen, areas of mucous membrane in the colon or in the lower part of the ileum are closely stippled with black dots, giving the "shaven beard" appearance, probably due to the deposit of pigment perhaps from minute blood extravasations in the small glands or in the superficial part of the mucous membrane.

Microscopic examination of the intestine may show well-marked lesions even where there is nothing abnormal to the naked eye. The earliest change is a round-cell infiltration of the mucosa, with some shedding of the epithelium; in more advanced cases the sub-mucosa is also infiltrated, the vessels are distended, and there is distinct increase in size of the solitary follicles and of the Peyer's patches from similar round-cell infiltration. Sometimes the mucosa is found at some parts to have disappeared over a collection of small round cells which form the base of an ulcer. In the most severe cases there may be considerable exudation of fibrin together with the round-cell infiltration, and the presence of such fibrin on the surface of the mucosa or over an ulcerated area produces the membrane which is seen in some of these severe cases.

In chronic diarrhœa similar changes occur, but repair has already taken place to some extent, the inflammatory infiltration has become organised, and a newly-formed fibrous tissue is seen separating and compressing the glands of the mucous membrane, which have in this way disappeared. An actual cirrhosis of the mucous membrane thus takes place, and the affected areas are no longer able to absorb properly. These histological changes account for the intractable wasting which may follow a chronic diarrhœa.

Treatment.—(*a*) Prophylactic.—The first and most important of all measures is the continuance of breast-feeding, and breast alone, until the age of at least nine months, and it is wise to prolong it for two or three months longer if in this way weaning during the hot summer months, especially July and August, can be avoided.

In hand-fed infants strict care in diet (*vide* "Infant Feeding"), and in particular the sterilisation or pasteurisation of the milk, will do much to avert the risk of diarrhœa. The use of the old-fashioned feeding-bottle, with its long rubber tube coated inside with stale milk and bacteria, is to be strictly forbidden, and the modern boat-shaped bottle substituted.

There can be no doubt that attention to the sanitary arrangements of the house, and in particular of the nursery, is of the greatest importance. Some of the worst cases of diarrhœa in infants and in young children occur where sanitation is in some way defective, it may be from some leakage of a soil pipe, or from defective traps in a water-closet outside their room. Ventilation must be free; soiled diapers must never be allowed to remain in the nursery. Town children may with advantage be sent away to the country or seaside during the hot season; the cooler and purer air of the country certainly has a considerable value as a curative agent when diarrhœa is already present, and probably is also of great value in prophylaxis.

The influence of chill in the production of diarrhœa in infants must not be forgotten. Knitted drawers worn over the diaper should cover the abdomen and the upper part of the thighs, and when the child goes out woollen gaiters reaching well up the thighs, or better still woollen drawers reaching down to the ankles, should be worn, not only on winter days, but also on the chilly days of autumn and spring.

(*b*) Therapeutic.—In the treatment of diarrhœa in infancy diet is often of greater importance than drugs. In a large number of cases, especially of chronic diarrhœa, the feeding is found to be entirely unsuitable, and the first step, sometimes indeed the only one necessary, is to correct the diet in accordance with the age of the infant. It will often happen, however, that some special diet is required as a temporary measure. In most cases of acute diarrhœa milk must be omitted altogether for some days; but where it is thought advisable to continue it, the milk should be well diluted, and lime water should be freely given with it, for, apart from its value in increasing the digestibility of the

milk, lime water itself certainly has some controlling influence on the diarrhœa. Rice water may be used as a diluent; barley water seems in some cases, especially perhaps in the younger infants, to keep up the diarrhœa, sometimes, however, it agrees well, and may be used alone for a day or two where milk cannot be given.

Broths are very useful, particularly veal broth and chicken broth; mutton broth can be used where these cannot be obtained. Fat must be removed from the meat before the broth is made, and any fat in the broth must be strained off. Beaf tea is better avoided; it is apt to make the diarrhœa worse and the stools offensive. Raw meat juice is sometimes useful, but rather as an alternative with some other food, such as whey or barley water, than alone. Whey is particularly valuable where there is much vomiting (*vide* p. 408). In infants beyond the age of one year, and particularly in the more chronic cases of diarrhœa, thin arrowroot is valuable, and seems to aid in checking the disorder.

In very acute cases of diarrhœa, especially where there is much vomiting, and in choleraic diarrhœa, the drain of fluid from the child is very rapid and severe, and appears in many cases to be in part, at least, the actual cause of death. The immediate supply of fluid in such cases is a vital necessity, and the quantity is of much more importance than the quality. If fluid with some nutritive value, such as barley water or albumin water, can be given by mouth and retained, so much the better, but if not, plain sterilised water is almost as useful; and if even water cannot be retained when given by the mouth, or cannot be taken in sufficient quantities, it must be given either by rectum, a method not always available owing to the diarrhœa, or subcutaneously by several injections in various parts of the body. Intravenous injection is not an easy procedure in infants, and as the injections may have to be repeated several times it is simpler and better to inject the water hypodermically. Albumin water has been used for hypodermic injection, but this is unnecessary and involves some risk of inflammation. It is unnecessary even to use a saline solution, simple boiled water seems to serve equally well; as much as an ounce and a half or two ounces can be injected altogether, and the injection repeated in a few hours' time if necessary. Intraperitoneal injection of saline solution cannot be recommended; even with the most scrupulous care it is not free from serious risks, and although it has caused decided improvement within the writer's experience, it has little if any advantage over the much safer method of hypodermic injection.

Whatever method of feeding be adopted, the presence of vomiting in severe cases usually makes it necessary to give the food in small quantities at short intervals; and all food given by the mouth should be either cold or only just warm.

In almost all cases of acute diarrhœa, where there is much exhaustion, stimulants are necessary. In severe cases where there is collapse nothing is more effectual than a hot mustard bath. But unless this is given skilfully it may do more harm than good. Too often one has seen infants held with their backs under water, while the whole of the front of the chest and parts of the limbs are projecting wet above the surface of the water, simply getting chilled by evaporation. The water must be deep enough to cover the child completely up to its neck; one brimming tablespoonful of mustard (or more in proportion to the amount of water) should be mixed into thin cream with tepid water and then stirred into a gallon of water at a temperature of 100°; the infant should not be kept in the bath more than three minutes, and should then be taken out and immediately wrapped in a hot blanket, while it is rapidly dried with a hot towel without any exposure. It must then be put back to bed in a hot blanket with hot bottles.

Sometimes it is more convenient to use a mustard pack, the infant being wrapped for about ten minutes in a sheet wrung out of mustard and water in the proportion of 1-2 tablespoonfuls of mustard to the gallon.

Brandy or whisky should be given internally; in severe cases 10-15 drops may be given in a teaspoonful of water as often as every two hours to an infant three months old, and for an infant a year old as much as an ounce in the twenty-four hours may be necessary. A drop of sal volatile given occasionally in a teaspoonful of the food not only acts as a stimulant, but sometimes checks vomiting; it must not, however, be used too freely, as it may aggravate the diarrhœa.

An extremely useful stimulant where exhaustion threatens to prove fatal is strychnine administered hypodermically; $\frac{1}{4}$ minim of the liquor may be used for an infant three months old, and may be repeated after three or four hours. As a last resource, last because painful, brandy or ether (♏x.-xx.) may be injected subcutaneously.

In simple diarrhœa a single aperient dose of castor oil or of calomel may be required to clear out the offending substance, and this alone may be sufficient; but usually medical advice is not sought until the bowels have already been open so freely that the irritant has probably already been removed, and sedatives are needed to allay the resulting catarrh. For this purpose opium is the most generally useful drug; the tincture may be given in doses of ♏$\frac{1}{8}$-$\frac{1}{4}$ three times a day to an infant four months old, ♏$\frac{1}{2}$ three times a day at six months, and ♏j. every four hours at twelve months. Bismuth is often useful with opium or alone, but it must be given freely; five grains is a suitable dose for an infant a year old; it is perhaps best given in sus-

pension, with sodium bicarbonate. Astringents are sometimes useful in such cases after the acute symptoms have passed off. Some such mixture as the following may be tried :—Tr. catechu ♏x., Spirit. chloroformi ♏ij., Mist. cretæ ad ʒj.; or a mixture containing hæmatoxylum, or some preparation of tannic acid, may be given. But the results of such astringents in any form of infantile diarrhœa are disappointing; in many cases they do not seem to have the least effect. A combination of acid. sulph. dil. or acid. nitric. dil. (♏ij.-iv.) with opium often does more good.

If the diarrhœa does not cease after a few days the following mixture may be found effectual.—Ol. ricini ♏v., Mucilag. acaciæ ♏xv., Aq. menth. pip. ad ʒj. There are few drugs which are more generally useful in the treatment of subacute or chronic diarrhœa than castor oil given in this way, especially if a small dose of opium, or, where the stools are offensive, a drop of liquor hydrargyri perchloridi, be added to it.

In many of the more severe cases of inflammatory diarrhœa, especially where the stools are green and offensive, grey powder is of great value, and often does much good in combination with Dover's powder; thus to an infant of six months a quarter of a grain of each may be given three times a day.

As to the value of antiseptics other than mercury there is some difference of opinion. In the most acute cases of summer diarrhœa the writer has not found them of much use; but in the less acute cases, and still more when the diarrhœa has become chronic, they are sometimes helpful. Perhaps the most useful of these drugs are the salicylates: salicylate of soda (gr. ij.-iv.) especially in combination with bismuth, or bismuth salicylate itself, has certainly done good in many cases, and these preparations have seemed on the whole more effectual than salol (gr. j.-iij.). Naphthalin (gr. j.-iij.), β-naphthol (gr. j.-iij.), glycerin acidi carbolici (♏j.-ij.), and resorcin (gr. ij.-iv.) are all worthy of trial, and have been praised by various observers. The last mentioned is the most convenient, for not only is it readily soluble in water, but it is less disagreeable in taste than the others.

Creasote is specially valuable where there is much tendency to abdominal distension, a condition which often goes with frothy liquid stools; it may be given in half-minim doses every three or four hours with the castor oil mixture mentioned above. In cases where from the passage of mucus and blood, or from tenderness along the course of the colon, there is reason to believe that the colon is chiefly affected, irrigation of the colon may be of use. This may be done once or twice a day with plain warm water or with a solution of boracic acid, or the addition of tannic acid (gr. xx.-xxx. to the pint) may be tried. It is useful after the irrigation to give a small enema of starch with tincture of opium (♏j.-iij.), and such an enema should be given in any case where great frequency of stools with much straining or prolapse of the rectum indicates excessive irritability of the lower part of the intestine. Occasionally irrigation seems to be of value in cases where there is no evidence of special affection of the colon; in acute cases of diarrhœa it has at any rate the recommendation that a certain amount of the fluid is likely to be absorbed.

In choleraic cases the vomiting is generally as serious as the diarrhœa, and often it is almost useless to administer drugs by the mouth. Good results have been obtained by washing out the stomach in such cases; and a mustard poultice to the epigastrium may help to check the vomiting. If opium can be retained it is well to give a dose of opium with brandy at first, and then bismuth may be tried. Frequent minute doses of grey powder have seemed to do good. Dr. Eustace Smith recommends a sixth of a grain of calomel every half-hour. In infants of twelve months or more hypodermic injections of morphia (gr. $\frac{1}{40}$ for a child of twelve months) have done good. Usually, however, these cases are so acute, and prostration is so rapid, that the most important part of treatment is free stimulation by one or other of the methods described above.

Chronic diarrhœa generally calls for dietetic treatment chiefly; but some drugs are extremely useful in this condition, and none more so than the castor oil mixture mentioned above. Antiseptics also, as already pointed out, are of more service in chronic than acute conditions. The combination of grey powder with Dover's powder is specially useful, and may be given with advantage in addition to the castor oil mixture. The astringents are generally useless after a diarrhœa has already continued many weeks.

Lienteric Diarrhœa.—It is convenient to mention this disorder here although it occurs rather in children four to ten years of age than in infants. It differs from the forms of diarrhœa described above, inasmuch as it is quite independent of errors of diet, of infection, and of seasons. It is probably closely allied to the diarrhœa which occurs sometimes both in children and in adults as the result of fright or emotion. In a considerable proportion of these cases there is a family history of rheumatism, and the occurrence of lienteric diarrhœa in such cases is a manifestation of the nervous temperament which is so often present in the children of rheumatic families.

The characteristic symptom of this condition is the evacuation of the bowels immediately after every meal; sometimes, indeed, the desire to defecate comes on before the meal is finished. The motions are not necessarily loose, but often contain undigested food; as the mother says,

"directly the child eats anything it goes through him." Sometimes there is slight colicky pain before the bowels are open, but often this is absent. Nutrition gradually suffers, but not to a very marked degree. There can be little doubt that the condition is really a functional neurosis; the intestine shows undue reflex excitability, so that the taking of food into the stomach immediately starts peristalsis of the bowel. Often when the child is brought for medical advice the condition has already lasted several weeks or months, but improvement is generally rapid under suitable treatment.

Treatment.—A mixture of bromides with belladonna given an hour before each meal is very effectual. Small doses of Dover's powder may be used similarly. When the frequency of the evacuations has diminished, liquor arsenicalis (♏ j.-iij.) should be substituted, and may usefully be given with nux vomica.

Constipation

Both in breast-fed and hand-fed infants constipation is a very common trouble, and often extremely difficult to remedy. In breast-fed infants the cause is usually some defect in the mother's milk, particularly deficiency of fat. Sometimes no fault whatever can be found in the milk, and it seems probable that the trouble is due simply to feebleness of peristalsis, either as part of the general weakness of infancy, or as a congenital condition perhaps the result of defective innervation. It is probable also that the sharp kinks which are so often present with the looping of the long sigmoid flexure in infancy (*vide* p. 401) may hinder the progress of the fæces, and the accumulation of fæces in turn aggravates the kink, so that a vicious circle is established which may account for the obstinate constipation which is not infrequent in infants

In hand-fed infants, apart from congenital causes, the diet is usually at fault. The mixtures of milk and water which mothers usually give their babies are very deficient in fat, the patent foods are even more so. Sometimes excess of casein seems to cause constipation, but in such cases there is often some catarrh of the bowel, and attacks of diarrhœa may alternate with constipation. A chronic catarrhal condition, such as is common in rickets, is not infrequently a cause of constipation in hand-fed infants, but the rickety constipation is probably due also in part to muscular weakness. Where the stools are pale and whitish the constipation seems to be due to deficiency of bile. Fissure at the anus is said to cause fear of defecation, and so constipation in some cases. Narrowing at the anus or rectum, as a congenital condition, may cause partial obstruction; and more acute obstruction from hernia or from intussusception may occur at any age. As a reflex condition constipation is sometimes a prominent and early symptom of cerebral disease, particularly of meningitis.

Symptoms.—Infants who are costive are often miserable and fretful, they sleep badly, and occasionally strain and grunt; flatulence and colic also are commonly associated with constipation; the temperature may be slightly raised; and where constipation is habitual, nutrition suffers and growth may be delayed. Infants will sometimes strain violently, and quite exhaust themselves in their efforts to defecate, and when the firm fæces are passed they are found to be streaked with blood. As direct results of the straining prolapsus ani and hernia may occur. In two cases under the writer's care piles were present at one month and at eighteen months of age respectively.

Treatment.—This must depend on the cause. If the mother's milk be suspected of poorness she should be encouraged to drink milk freely, and to take a liberal diet with plenty of milk food; it is well if she can take also malt or cod-liver oil. The treatment of the child by drugs given to the mother is usually unsatisfactory, but any of the saline aperients can be used in this way. In hand-fed children the feeding generally requires modification in the direction of additional cream or of diminution—rarely increase—of casein. The addition of a teaspoonful of Mellin's food to one or two of the meals, or the use of malt extract instead of sugar for sweetening, or a little manna (gr. x.-xxx.) given in the milk two or three times a day, may be successful. Sometimes fine oatmeal made into a cream and added to the milk for breakfast, or in infants of ten months or more a drink of beef tea, may be sufficiently laxative. To infants of eighteen months or more a little of the juice of stewed prunes may be given, or well-baked apples; sometimes a little treacle given with oatmeal, or a little honey, will keep the bowels regular. Gentle massage along the course of the colon should be done every morning, and it is most important that the infant should be held over a chamber at a fixed time once or twice a day, even if the bowels fail to act, for in infants as in adults "habit is second nature."

Drugs may be necessary, but the less they are used the better. Saline aperients, sodium phosphate (gr. x.-xv.), magnesium sulphate (gr. v.-x), or sodium sulphate (gr. ij.-iv.) may be used alone or in combination; sometimes a single dose in the morning is sufficient, sometimes they must be given three times a day. Syrupus cascaræ aromaticus (♏ v.-x.) with sodium sulphate may be tried, but cascara sometimes gripes, and is very unreliable in its action. Where the constipation seems to be due to weakness, minute doses of nux vomica with compound decoction of aloes may be tried. Sometimes a small dose of grey powder given once or twice a day is effectual. Where the stools are pale or putty-coloured, tincture of

podophyllum (♏ j.-ij. three times a day) is sometimes serviceable; it may be given in mixture or on powdered sugar. As an occasional aperient, syrup of senna (♏ x.-xv.) or calomel (gr. j.-ji.) is suitable for infants; castor oil should not be used where there is any tendency to habitual constipation, as it tends to make this worse afterwards.

Enemata are often necessary, and for occasional use there is no objection to soap and water or glycerin, but the smallest that will work is the best, for it is possible that large injections may produce some atony of the gut, and so aggravate the constipation. On this account olive oil ($\frac{1}{2}$-1 ounce), followed by one or two ounces of soap and water, is better than a large soap and water enema. Where frequent injections are necessary, glycerin is apt to be too irritating to the rectum. The simplest and safest remedy for frequent use is a piece of soap used as a suppository, and for a time this is usually efficient, but if used very often it is apt to lose its effect.

Congenital Dilatation of the Colon

This name has been applied to a rare condition in which obstinate constipation is associated with great dilatation and hypertrophy of the colon, apparently without organic obstruction. The name is perhaps not strictly accurate, for although it seems probable that some congenital abnormality of function or structure underlies this condition, and the chief symptom, namely, constipation, has in most cases been present from birth, the dilatation of the colon cannot generally be detected clinically until some weeks or even months later.

Almost all the cases of "idiopathic" dilatation of the colon which occur in childhood appear to be of this variety, but a very similar condition occasionally begins in adult life, although in some of these cases also there is a history of much constipation from birth.

It seems possible that there may be some family predisposition to this condition, for in one case that came under the writer's observation a brother had died of it; and in another a second child had died apparently with the same condition.

It is a curious fact that almost all the recorded cases have been in boys.

Symptoms.—The child is apparently healthy at birth, but there is great difficulty in getting the bowels open. For a week or more they may not act, and in one case nineteen days elapsed after birth before they were opened. Very rarely constipation does not begin until some weeks or even for two or three months after birth. When the child is a few months old, sometimes only when it is a few days old, the abdomen is noticed to be large, and with increasing constipation the abdomen may become enormously distended. In one case the distension was so great that the heart was pushed up so that the upper limit of cardiac dulness was at the first rib. Œdema of the lower limbs may result from pressure of the distended bowel on the veins.

The most characteristic feature, however, is the visible peristalsis of the colon, which has sometimes attracted even the mother's notice. The surface of the abdomen is seen to rise and fall in irregular eminences and depressions from time to time, corresponding with the slow vermicular movements of the enormous coils of hypertrophied colon which seem to fill the whole abdomen.

Usually in spite of all treatment the constipation persists; the bowels are open only at long intervals, and by artificial means; in two cases under the writer's observation the intervals were sometimes as long as five or six weeks. During these periods there is often headache and vomiting, and the breath is sometimes very offensive. Abdominal pain is usually absent throughout, except sometimes towards the end of the illness.

The appetite in some of these cases is very large, but the child does not thrive; it is poorly nourished, and in the latter stages may be considerably wasted.

Sooner or later, sometimes within the first year, sometimes not till the child is ten or twelve years old, the distension of the abdomen becomes extreme, there is some tenderness on palpation, and the constipation is replaced by diarrhœa, the stools being liquid, drab-coloured, or pale yellow, and perhaps frothy. The temperature, which hitherto has been normal, may now rise, and in a few days, partly from respiratory difficulty, owing to the abdominal distension, partly perhaps poisoned by absorption from the dilated intestine, the child dies.

Morbid Anatomy and Pathology.—The large intestine is found to be enormously dilated, and its muscular wall hypertrophied. The dilatation is not always uniform throughout, and it probably varies somewhat during life with the changing relations of the bowel in peristalsis; the hypertrophy, however, seems to affect the whole large intestine down to the anus. Often some recent ulceration of the mucous membrane is present, generally transverse and linear in appearance, the result, no doubt, of the acute and extreme distension which precedes death; the ulceration is seldom extensive, and may be limited to the part where the distension is most marked. The distension of the bowl is found to be due partly to gas, partly to accumulation of fæces, which are often quite liquid; but the condition found post-mortem is probably different from that which exists during the greater part of life, for during the last few days or weeks of life, as already mentioned, acute symptoms supervene, and there can be little doubt that these coincide

with the occurrence of a superadded acute atonic distension of the bowel, and inflammation and ulceration of the mucous membrane; the pale liquid fæces also are a symptom of this final exacerbation. The small intestine is normal. The most remarkable characteristic in the morbid anatomy of this condition is the apparent absence of any mechanical obstruction. Its pathology is uncertain. It is noteworthy that there is no evidence at present that there is any hypertrophy or dilatation of the colon at birth, the only part of the condition which is actually congenital is the constipation. Any theory which is to explain all the cases must account for the fact that in some cases, at least, the whole large intestine, down to the anus, is hypertrophied. Spasm of the rectum or of the sphincter ani might explain this, but in most cases no such spasm has been found. Kinking at the sigmoid loop, or some abnormal arrangement of the mesentery, fails to explain the affection of the part below the sigmoid flexure. Possibly in some cases there is some congenital defect of innervation of the intestine so that its contractions are ineffectual and constipation results, with secondary dilatation and hypertrophy.

Diagnosis.—The salient points in the diagnosis are the history of constipation from or soon after birth, the presence of visible peristalsis of greatly enlarged coils of intestine, and the absence of any obvious cause for obstruction. This last point is important, for a congenital stenosis of the rectum, or a narrowing of the anus, either congenitally or from an imperfect result after operation for imperforate anus, may produce an almost exactly similar result. Such malformations are readily excluded by examination.

This condition has been mistaken for tuberculous peritonitis; and in the later stage the thin child with its distended abdomen suggests such a diagnosis at first sight; but apart from the history and the dilated coils of intestine, the congenital condition is distinguished by the complete absence of that doughy resistance and the band-like masses which are so characteristic on palpation of the abdomen in tuberculous peritonitis.

The absence of fluid thrill and of the characteristic shifting of dulness with position, taken together with the history, should serve to exclude ascites.

Very rarely in childhood adhesions from past peritonitis cause obstruction of the bowels with distension of the abdomen and visible peristalsis of dilated coils of intestine, but such adhesions are likely to affect the small intestine, and careful inspection will generally detect this difference partly from the size and shape and partly from the position of the coils. Too much importance, however, must not be attached to position, for in congenital dilatation of the colon the huge coils occupy most unusual positions, the sigmoid loop in particular frequently extends across into the right iliac fossa, and may rise above the umbilicus.

Prognosis.—In five cases under the writer's observation the ages at death were respectively 4½ months, 4¾ months, 5 months, 3 years, and 3¾ years, and two others were still living at the ages of 7 years and 10 years. Two cases have been recorded in which the patients survived to adult life, but this is quite exceptional. In almost all the recorded cases death has occurred under the age of 12 years. Whether the condition is ever recoverable may be doubted; certainly under treatment there may be considerable improvement.

Treatment.—In the early stage much can be done to prevent the condition becoming worse. It must be impressed on the parents how important it is that they should never relax their efforts to keep the child's bowels regular. The bowels must be opened at least every two days.

As to the means to be employed to overcome the constipation, careful selection is necessary. Drugs given by mouth too often prove useless, but are worthy of trial, especially in the early stage, before the dilatation is very great. A mixture of liquor strychninæ with sodium and magnesium sulphate may be given three times a day, or belladonna with aloes and nux vomica may be tried. A morning dose of apenta water has acted well. In some cases calomel has seemed to be most useful, and probably even in the later stages has a decided value, not only as an aperient, but also as an antiseptic, where there is a tendency to decomposition and fermentation in the dilated bowel.

Gentle massage of the colon along its course is quite safe, and sometimes decidedly beneficial, in the early stage, but when distension is extreme, especially when the stools are liquid and there is tenderness of the abdomen, the likelihood of ulceration must be borne in mind, for there may be, as Dr. Rolleston and Mr. Haward have suggested, a risk of rupturing the intestine by massage.

In almost all cases drugs given by mouth prove ineffectual sooner or later, and enemata become necessary. For the reasons stated above (*vide* p. 419), olive oil, either alone or mixed with castor oil, followed at once by a small soap enema, is perhaps the least open to objection. In some cases it becomes necessary to clear out the accumulated fæces from the rectum with the finger or with a scoop.

The diet in the early stage may be modified, as in cases of simple constipation; in the later stage it is well to let the diet be as simple as possible, and those articles are to be preferred which leave but little fæcal residue, such as milk, eggs, soup, etc.

At any time an acute exacerbation of distension is liable to occur, especially when the

bowels have been allowed to remain constipated for a long period. The likelihood of ulceration at such times must be remembered, and immediate steps must be taken to reduce the distension. Sometimes a turpentine enemata (Ol. terebinth ♏x., with soap and water ℥vj., for a child of six months) will give temporary relief. Sometimes mere digital examination of the rectum will allow gas and fluid fæces to escape. The passage of a long soft catheter up the rectum may be tried, and, combined with very gentle pressure on the colon, this may evacuate some of the gas. If gas or liquid fæces continue to come away through the tube in occasional gushes it is sometimes well to leave the tube in the rectum for an hour or more to prevent reaccumulation of flatus. Creasote (♏$\frac{1}{4}$-j. every three hours) should be given by mouth, as it certainly gives some relief to acute distension.

The question of surgical interference is one of some difficulty. In the early stage the general health is often quite good, and one has to balance the possibility of the child living for several years, perhaps even to adult life, with the use of simple medicinal remedies, against a possible cure by an operation which experience shows to be one of great risk. On the other hand, if surgical interference be deferred until the distension is extreme, the child is in far worse condition to stand the operation, and the difficulties of operation are considerably increased. Some of the cases in which surgical treatment has been tried were in the extreme stage, and the fatal results are perhaps to be attributed in part to this fact. It must be mentioned, however, that in two cases operation appears to have been successful, one in which colotomy was performed (Osler), the other in which the descending colon and rectum were resected (Treves).

Prolapsus Recti

This is a frequent result of straining from any cause. It is more common in children of two to six years than in actual infancy. In infants it is often associated with a marasmic condition, the tissues of the ischio-rectal fossæ are so wasted that they afford insufficient support to the rectum, which therefore tends to prolapse. Constipation, diarrhœa, the irritation of thread-worms, a polypus in the rectum, a tight foreskin, calculus in the bladder—any of these conditions may be the exciting cause of prolapse of the rectum.

Symptoms.—In the mildest cases only a narrow rim of mucous membrane projects through the anus, and the projection may be so slight that at the end of defecation it is pulled in by muscular contraction without artificial aid. Such a degree is of importance only as the precursor of the more troublesome condition, where the prolapsed mucous membrane remains down until returned artificially. In more severe cases two or three inches of extroverted rectum may project from the anus, and in this condition the whole wall of the bowel descends, and even the peritoneal pouch may be dragged down. The prolapsed portion is seen as a cylindrical tumour projecting from the anus, and showing on its outside the bright red, often congested, mucous membrane, and at its apex the lumen of the bowel. Where the prolapse has been allowed to remain down for several hours or days the exposed mucous membrane may become dry and cracked, bleeding on the least manipulation, and sometimes deeply ulcerated.

Prognosis.—As a rule, treatment of the cause produces rapid improvement, the prolapse becomes less frequent, and then ceases altogether. So long as it can be reduced easily, and is never allowed to remain down more than a few minutes, the prolapse is not dangerous; but where it has been allowed to remain unreduced for several days, and reduction has been accomplished with great difficulty, the writer has twice known the condition to prove fatal.

Treatment.—The reduction of the prolapse can usually be accomplished by gentle pressure with the fingers covered by a soft oiled rag, the child meanwhile lying on its side. Application of cold, in the form of iced water, to the prolapsed mucous membrane may assist reduction in difficult cases. Where there is great difficulty, owing to continual straining, or to swelling or inflammation of a neglected prolapse, it may be necessary to administer chloroform. After the prolapse has been returned a starch and opium enema should be given, and for some days astringent injections, or injections of cold water, may be used once or twice a day. The application of a broad strap of plaster across the buttocks, so as to pull the buttocks together and support the rectum, is often effectual, and with this a pad over the anus may be combined. Merely lying on the back seems to do good in some cases. A very efficient method for bad cases is to suspend the legs by stirrups, as is done sometimes for fractured thigh, the child lying on its back with the hips supported on a pillow. In a troublesome case the child should defecate, lying either on its back or on its side, and the buttocks may be supported during defecation by the pressure of a hand placed on either side of the anus. It has been recommended that a chamber-pot with a wooden lid, in which there is the smallest practicable opening, should be used, the lid pressing on the buttocks and so supporting the rectum.

Operative measures are hardly ever necessary for prolapse in childhood. In some cases, however, where it has persistently recurred, the laxity of mucous membrane which predisposes to recurrence has been treated by the actual cautery; this is drawn in three or four vertical

lines down the whole length of the mucous membrane on the exposed surface of the prolapsed portion. The application of acid nitrate of mercury has also been recommended (Jacobson). Quite as important as the local treatment is the treatment of the constipation or diarrhœa or other cause to which the prolapse is due; in many cases, indeed, this is all that is required.

Gastro-jejunostomy.—An operation by which an artificial communication is established between the stomach and the jejunum; gastronesteostomy.

Gastrolysis.—An operation in which the abdomen is opened for the purpose of separating perigastric adhesions. *See* Stomach, Surgical Affections (*Gastric Ulcer, Complications, Treatment*).

Gastromalacia. — A post - mortem softening or digestion of the gastric mucous membrane, once regarded as an ante-mortem change. *See* Gastro-Intestinal Disorders of Infancy (*Diseases of the Stomach and Intestine, Softening of the Stomach*).

Gastromelus.—A teratological type in which a supernumerary limb is found attached to the abdomen. *See* Teratology.

Gastro - parasitus. — A group of double monsters (disomata) of the asymmetrical type in which the one twin (usually very imperfect) is attached as a parasite to the abdomen of the other, or lies within its peritoneal cavity (*fœtus in fœtu*). *See* Teratology.

Gastropexy. — An operation in which the abdomen is opened for the purpose of shortening the superior attachments of the stomach, as in gastroptosis. *See* Stomach, Surgical Affections (*Operations*).

Gastroplasty.—An operation performed for the relief of hour-glass stomach. *See* Stomach, Surgical Affections (*Hour-Glass Stomach, Operations*).

Gastroplication. — An operation by which artificial folds are made in the walls of the stomach to diminish the size of the cavity of the viscus; it is performed in cases of dilated stomach, with or without gastroptosis. *See* Stomach and Duodenum, Diseases of (*Gastroectasis, Treatment*); Stomach, Surgical Affections (*Gastroectasis, Operations*).

Gastroptosis. — Downward displacement of the stomach, often associated with enteroptosis. *See* Stomach and Duodenum, Diseases of (*Morbid Anatomy, Gastroptosis*); Stomach, Surgical Affections (*Gastroptosis, Operations*); Enteroptosis (*Diagnosis*); Indigestion (*Symptoms, Sensory*).

Gastrorrhagia. — Hæmorrhage from the stomach. *See* Hæmatemesis; Stomach, Surgical Affections of (*Gastric Ulcer, Complications, Hœmorrhage, Operation*).

Gastrorrhaphy. — The closure of wounds of the abdomen or of the stomach, or (more recently) of a perforated gastric ulcer. *See* Stomach, Surgical Affections (*Operations*).

Gastrorrhexis. — Rupture of the stomach.

Gastroschisis.—Defect of the abdominal walls with protrusion of the viscera. *See* Teratology.

Gastrospasmus. — Cramp of the stomach.

Gastrostomy.—The establishment of a gastric fistula, as in cases of malignant or innocent stricture of the œsophagus, and in some instances of recent injuries to the œsophagus. *See* Stomach, Surgical Affections of (*Operations*); Œsophagus, Injuries and Diseases of (*Growths, Malignant, Treatment*).

Gastrotomy.—The operation of opening into the stomach by abdominal section and incision of the gastric wall, *e.g.* for the removal of a foreign body; the term was formerly employed in the general sense of opening into the abdominal cavity, *e.g.* for ruptured uterus.

Gastroxynsis.—Periodical supersecretion of gastric juice, occurring in nervous dyspepsia, especially in cases of neurasthenia and locomotor ataxia (*Rossbach*).

Gastrula.—When the single-walled blastula of germinal life becomes a double-walled sac (consisting of ectoderm and endoderm), it is named a gastrula; its cavity is the archenteron, and the opening by which this cavity communicates with the exterior is the blastopore.

Gaultheria. — The oil of wintergreen (Gaultheriæ Oleum) contains a large quantity of methyl salicylate, which gives it its medicinal properties; it is given in doses of from 3 to 10 m. for the same purposes as the salicylates; and it is included in the Indian and Colonial Addendum (1900) to the British Pharmacopœia of 1898.

Gauze. *See* Bandages.

Gavage.—The artificial feeding of infants (*e.g.* those with cleft palate or inability to swallow) by means of a stomach tube; milk is the liquid usually given, and may be that of the mother of the child or that of the cow.

Gelasmus.—Spasmodic laughter, usually hysterical in nature. *See* Hysteria.

Gelatin.—Collagen, a non-elastic substance allied to the proteids, when boiled, takes up water to form gelatin, which is soluble in hot water but forms a jelly on cooling; it has a restricted value as a food stuff (it yields energy during its decomposition into urea), being acted upon by trypsin. The *Gelatinum* of the Pharmacopœia is made by boiling skin, tendons, and bones, and is prepared in sheets; it is used for making suppositories, medicated pessaries, capsules, lozenges, and for coating pills. In Bacteriology, it serves as a culture medium. Recently gelatin has been used in the treatment of hæmorrhages of different kinds (hæmoptysis, gastric and intestinal bleeding, hæmophilia, purpura, etc.) by subcutaneous and intra-venous injection, by the mouth or by the rectum; the gelatin so used must be sterilised (*e.g.* Merck's *gelatina sterilisata*).

Gelatinous Degeneration. *See* Joints, Diseases of (*Tuberculous Disease, Pathological Anatomy*).

Gelsemii Radix.—The dried rhizome and roots of "yellow jasmine"—*Gelsemium nitidum*, imported from Carolina. *Dose*—5-15 grs. Its constituents are—1. *Gelseminine*, a powerful alkaloid in the form of minute yellowish crystals, sparingly soluble in water and freely soluble in alcohol, ether, and dilute acids. *Dose*—$\frac{1}{100}$-$\frac{1}{32}$ gr. *Preparations*—(1) Gelseminінæ Hydrochloridum, freely soluble in water. *Dose*—$\frac{1}{60}$-$\frac{1}{20}$ gr.; (2) Lamellæ Gelseminінæ, each containing $\frac{1}{500}$ gr. of Gelseminine. 2. *Gelsemine*, a much less powerful alkaloid, which must be carefully distinguished from Gelseminine. *Dose*—$\frac{1}{2}$-2 grs. 3. *Gelsemic Acid.* The only official preparation of Gelsemium is *Tinctura Gelsemii. Dose*—5-15 m. On account of its powerful depressant effect on the anterior cornu of the spinal cord and the motor centres at the base of the brain, gelsemium was formerly used in the treatment of chorea, tetanus, acute mania, spasmodic asthma, and conditions accompanied by cerebral excitement. Its action in these diseases, however, is very uncertain, and sometimes even dangerous. It is now given almost solely for trigeminal neuralgia, frequently in combination with butyl chloral hydrate. It is apparently of little value in other forms of neuralgia. It paralyses accommodation, and dilates the pupil, and for this purpose it may be applied locally in the form of the lamella of Gelseminine.

Gelsemine and **Gelseminine.** *See* Gelsemii Radix.

Gemelli Coaliti.—United twins; a double monster.

Gemelliparous.—Giving birth to twins; pluriparous.

Gemmules.—Little buds upon the course of the dendrites of nerve cells which perhaps serve to bring a neuron at one time into relation with one set of neurons, and at another time with another; formerly, the name was given to the small particles supposed by Darwin to be given off by all the tissues of the body to form the reproductive cell (ovum or spermatozoon), in order to give a physical basis for the theory of heredity.

Genal.—Relating to the cheeks.

General Paralysis.

See also Bedsores (*Clinical Types*); Brain, Tumours of (*Symptoms, Diagnosis*); Brain, Cystic Degeneration; Insanity, Pathology of; Mental Deficiency (*Developmental, Juvenile General Paralysis*); Nails, Affections of (*in General Diseases*); Osteo-Arthropathies (*in General Paralysis*); Paralysis (*Paralysis with Tremor or Ataxy*); Pregnancy, Affections and Complications (*Disorders of Nervous System*); Pupil (*Paradoxical Pupil*); Stomach and Duodenum (*Catarrhal Changes in General Paralysis*); Syphilis (*Hereditary Syphilis in Children*); Tabes Dorsalis (*Etiology, Diagnosis*); Ulcers and Ulceration (*Perforating Ulcer*); Unconsciousness (*Comatose Attacks*).

Synonyms.—Progressive general paralysis; Dementia paralytica; Paretic dementia; General paresis; General paralysis of the insane. *Paralysie générale des aliénés, Folie paralytique* (Fr.); *Paralyse des Irren* (Ger.).

Definition.—General paralysis of the insane is a progressive and incurable disease of the nervous system, characterised by bodily symptoms which end in paralysis, and mental symptoms which end in dementia, the disease running a fairly definite course of development, and terminating fatally in from two to three years.

History.—Willis is accredited with having first drawn attention to this disease as early as 1672. Haslam and Perfect also seem to have recognised it at the close of the last century. Esquirol noted the extreme gravity of those cases in which dementia was complicated with paralysis, but it was reserved for his pupils,

Georget, to describe the condition under the name of "chronic muscular paralysis," Delaye, under that of "incomplete general paralysis," and Calmeil, under that of "paralysis observed in the insane." Up to this time the affection was regarded as a special form of paralysis complicating an already existing mental disease. Bayle (1822) formulated a theory that the condition was not a mere complication of insanity, but a true morbid entity. He insisted on the existence of arachnitis or chronic meningitis as its predominant lesion, ambitious delusions as its necessary symptoms, and completed the picture by a description of its various stages of development.

Parchappe (1838) followed the lead and strove to prove that the affection was a special form of insanity. In 1846, however, there was a slight reaction, and Requin, MM. Sandras, Lunier, and Baillarger endeavoured to restrict this view. They recognised two forms, both progressive—the one with mental perversions, the other without. In 1858 a long discussion ensued between the partisans and opponents of Bayle's views, and the notion of the disease being a morbid entity was accepted. Since then, however, its anatomico-pathological features have been questioned, and an enormous mass of etiological and clinical data have gradually accumulated, with the result that the unitary theory is no longer able to hold its ground.

Ball described it "as a genus comprising many species," and at the present time many recognise that it is not a single malady, but a group of more or less distinct diseases, varying according to their causes or their lesions.

Even the most ardent of the advocates of the entity theory have in their descriptions of the disease included so many factors of causation, so varied symptoms, and so many different types of disease, that the olla podrida of general paralysis is in excess of that of any other disease.

The writer has pointed out (*Journ. of Mental Science*, p. 305, 1896) that before we can decide the question of the existence of true general paralysis, we have to differentiate between its group of symptoms and those of progressive partial paralysis. "From a pathological standpoint general paralysis, as we at present know it, seems to include every known cerebral degeneration as a possible adjunct of its pathology, *i.e.* it includes many affections which often, occurring independently, are in reality special paralyses. From the physical side we have to consider two broad types of disorder, viz. paralyses which are progressive and widespread, and paralyses which tend to progress, but which nevertheless remain, broadly speaking, only partial." From the mental side we have similar difficulties, inasmuch as the abnormal mental manifestations range through every grade of every faculty, thereby rendering a concise, comprehensive, or complete description impossible. The various new clinical facts, such as the remissions and the recognition of latent paralyses, syphilitic, saturnine, alcoholic, and paralytic insanities, have gradually overthrown the entity theory, which includes but fails to explain them. Were we to exclude all those types which simulate progressive general paralysis (pseudo forms), but which do not fulfil the rôle laid down in most text-books, general paralysis itself—as a distinct disease—would fade into insignificance, or even cease to exist. Until, however, we have carefully differentiated the innumerable factors included in what is at present known as "general paralysis," it is convenient to retain the term, and to use it in describing a paralytic dementia consisting essentially of dementia combined with progressive paralysis.

Causation.—General paralysis of the insane affects most commonly men of middle age whose nervous systems have been unduly exercised in the struggles and competitions of life, and in most cases the disease is further determined by vicious modes of life, errors of diet, injudicious use of stimulants, undue exercise of sexual functions, and neglect of the restorative power of sleep. It is comparatively rare in a purely agricultural population, but among those who dwell in cities and are subject to stress and strain it is very common. It is in the great centres of population, where the people are too thick on the ground, that the constant struggle for survival reacts most upon the nervous systems of a nervous age. It is among the full-bodied, vigorous, and active that the disease finds its abode, and it is the inextinguishable thirst for warm, vital human life, the preference for lit streets and the restless phantasmagoria of the city, that attracts men burning with the fires of a full life. The craving for sensations and satisfactions of many-sided life has its tragic side, and in endeavouring to devour the world men but consume themselves in the process. In brief, general paralysis is the outcome of stress and strain, alcohol and syphilis, all of which are met in the desire for keen, quick, ample life. In every sense it is the mind which is the standard of the man, and nothing succeeds like alcohol and syphilis in effacing the rational and moral faculties of even the noblest and richest.

It is comparatively seldom that alcohol alone is attributed as the cause of general paralysis, but intemperance does not necessarily mean only obvious and palpable drunkenness. From the very moment in which alcohol has disturbed the healthy exercise of the mental faculties, or has impaired the moral sense by unduly exciting the animal passions, or has in any way unfitted a person from discharging his duties in the proper struggle for survival, from that moment has there been guilt of intemperance. A nation must be depraved and wretched in

proportion as it abounds in drunkenness, and the ever-increasing weakness and brain decay as manifested in general paralysis is but one indication of the disastrous results of alcohol.

Of late years the relationship of syphilis to general paralysis has been much discussed. The writer's experience has led him to believe that syphilis is one of the most important of the factors of causation of a degenerative neurosis which is included in and described as general paralysis. At least 50 per cent of the cases of general paralysis under his care have had definite histories of syphilis, and probably, if all the facts were known, the percentage would be greater. The following types of insanity associated with syphilis occur:—

1. Types not entirely conforming to the classical descriptions of general paralysis.—The symptoms are: muscular tremors in tongue and hands, alterations in speech, inequalities of pupils, illusions or hallucinations of the special senses, depression or exaltation, loss of memory, mental weakness, temporary loss of consciousness, and not infrequently partial paralysis or hemiplegia. The diagnosis is difficult, and sometimes an autopsy alone is able to determine the existence of either simple cerebral syphilis or the diffuse periencephalitis of general paralysis. To this type Fournier has applied the name of *general syphilitic pseudo-paralysis*, or general pseudo-paralysis, implying in no respect any morbid entity whatever, still less a "modified general paralysis," and still less "general paralysis" peculiar to syphilis, but merely implying a particular modification of cerebral syphilis recalling more or less the pathological physiognomy of general paralysis.

2. Types which have a history of syphilis, but present no feature pertaining to syphilis, or are exclusively syphilitic, are not influenced by anti-syphilitic treatment, and have a pathology of the nature of a general encephalepathy rather than of cerebral syphilis. The symptoms are mainly those of so-called true general paralysis, and the condition may be termed *parasyphilitic general paralysis*.

3. Types seen in early life and due to inherited syphilis.—Not only may there be arrest or defect of mental or physical development, but there may be epilepsy. In some cases local lesions about the cranium, membranes, or the brain itself may account for the paralysis and the mental defect. There are, however, other cases in which the evidence of syphilis is quite clearly determined. These cases present a remarkable resemblance to general paralysis. The salient features of some of them are slow but progressive dementia, with steady development of generalised paralysis and great emaciation. Whether they are true cases of early general paralysis is still doubtful; they might be termed cases of *congenital parasyphilitic progressive paralysis*.

4. Numerous other types of cases with mental or physical symptoms occur, without, however, bearing any resemblance to progressive paralysis; they need not, therefore, be considered here.

The close relationship of tabes to general paralysis has been urged quite recently by Mott. The relative frequency of the occurrence of syphilis as a causal factor in both diseases is noticeable, but this alone hardly seems to justify the assumption that the two states are identical. Systemic spinal lesions of sensory or motor tracts might result from the same cause, and the proof of syphilitic origin of tabes, primary cerebral spastic paraplegia, or of general paralysis, does not warrant the assumption that they are one and the same disease.

Pathology.—None of the morbid brain changes found can be said to be distinctive or exclusive to general paralysis. The skull-cap is usually heavier than normal and may exhibit exostoses. The dura mater not uncommonly has rusty discoloration of its inner surface, or arachnoid cysts may be present. The subdural or subarachnoidal fluid is turbid and increased in amount. The arachnoid itself may be thickened, opaque, watery, or gelatinous. The pia, or stripping, adheres to the cortex cerebri along the summits of the gyri, especially of the frontal and parietal lobes. The gyri are atrophied and the lateral ventricles widened. The atrophy markedly affects the central and precentral gyri, basal ganglia, pons, and medulla. Small foci of softening are common in the grey and white matter of the parietal, central, and temporal gyri. Degeneration of the arterioles in conjunction with increase of arterial blood pressure may cause hæmorrhagic foci, especially about the basal ganglia, pons, and medulla, grey degeneration and atrophy of the cranial nerve roots are frequent. The dura mater of the spinal cord may be adherent to the vertebræ, the cord-tunics being thickened chiefly posteriorly. Hæmorrhagic or calcareous deposits on the inner surface are not so frequent as in the brain. Various systemic lesions of the cord itself occur, the blood-vessels are frequently congested, and adhesions may be formed between the pia and cortex.

The histological changes in the brain substances are—increase of blood-vessels, which are distended and tortuous; proliferation of nuclei of the vessel wall, and extravasation of leucocytes; blocking of the subadventitial and perivascular lymph spaces by lymph-corpuscles and waste products; increase of glia-cells, especially in the outermost layer of the cortex; degeneration of the nerve cells and their processes, beginning usually in the apical processes, the cell-body undergoing granular, fuscous, sclerotic changes, or vacuolation; wasting and degeneration of the cortical association nerve-fibres. In brief, the connective tissue becomes hypertrophied and the nerve tissues atrophied. The peripheral

nerves, the ganglia of the sympathetic, the muscles, and various organs of the body also exhibit evidences of degeneration. The real nature of the pathological process is as yet undetermined, and we do not know how far it may be attributed to the influence of a toxin derived from without or within.

Symptoms.—1. *Stadium Prodromale.*—Sometimes the disease comes on suddenly without any warning, but usually there are sensory, motor, or mental symptoms. The sensory warnings are, sudden losses or defects of the special senses, illusions, or hallucinations. The motor are, slight or temporary aphasia, hesitation in speech, loss of power of expression, blurring of articulation, tremor of tongue, ataxic or spastic alteration in gait, partial paralysis, convulsive seizures, inequality of pupils, reflex iridoplegia, changes in muscular electric reactions, alterations in handwriting, impairment of highest physical technique. The mental warnings are, loss of highest powers of adjustment of the mind to the requirements of the individual's profession or trade, loss of power of attention, restlessness, changes in temper, moral perversions, and loss of control. General instability, fitfulness, undue emotionalism, boastfulness, expansive benevolence, extravagance, etc. Vaso-motor symptoms, such as congestive attacks, cephalalgia, vertigo, hyperidrosis, local cutaneous hyperæmias, cyanosis, gastro-intestinal disorders may occur, or the trophic functions may be deranged, as shown by changes in the skin, hair, nails, muscles, and bones. The secretions may also be altered in their composition. This stadium has an average duration of one year.

2. *Stadium Acutum.*—Usually ushered in by melancholia, mania, dementia, or by convulsive seizures followed by confusion or stupor. The physical and mental symptoms become more fully developed. This stage usually extends over a period of about a year. The sensory symptoms become more marked in the form of definite illusions or hallucinations; the tinnitus aurium, deafness, diplopia, amblyopia, colour blindness, anosmia, ageusia, parageusia, and the disturbances of general sensation of the stadium prodromale, now become misinterpreted, and form a partial basis for the development of delusions. Melancholia with hypochondriasis may form the chief mental symptoms, or there may be a maniacal outburst with great exaltation, violence and delirium, with rapid advance towards terminal dementia and death. In milder types there may be merely weak-mindedness, with or without exaggerated ideas, or megalo-maniacal delusions. Impulsive acts and emotional outbursts are common. Alteration in character is very marked, the docile become irritable, and the hard facile. Seldom do they recognise the disease from which they suffer, even though they themselves may be medical men and able to diagnose it in others. The exaltation, or *megalomania*, though common in general paralysis, is not invariable nor is it confined to it. The general feeling of benevolence in general paralytics, however, is fairly constant, whereas it is not common in alcoholic mania, delusional insanity, or in monomanias of grandeur. Micromania, or the sense of "belittlement," also occurs, and is usually associated with some perversion of the cutaneous sense.

The *memory* may be altered in various ways. Sometimes in the earlier stages there is hypermnesia or exaltation of memory; in other cases there is a progressive amnesia from the first. The "law of regression" is here exemplified, in that the loss advances progressively from the unstable and most recent to the stable or more organised acquirements. In these cases of general dissolution of the memory an invariable path is followed, viz. memory of recent events goes first, then that of ideas in general, next feelings, and lastly acts. In instances of partial dissolution the loss also follows an invariable path, viz. proper names, common nouns, adjectives and verbs, interjections, gestures. Clinically, it is important to note that progressive loss of memory is usually pathognomonic of cerebral degeneration. In the early stages of general paralysis the impairment of memory is sometimes the most marked symptom. Sometimes the law of regression undergoes a remission, or the progression may be arrested for a time. Progressive loss of memory, however, is not confined to general paralysis, but may exist in syphilitic disease of the brain or meninges; various organic lesions of the brain, meninges, or vessels; in association with idiopathic morbid processes, traumatisms, toxic agents, etc. Sometimes progressive loss follows upon spinal affections, such as locomotor ataxy or multiple sclerosis; or it may result from epilepsy, hysteria, somnambulism, chorea, paralysis agitans, asthma, exophthalmic goitre, or myxœdema. Various forms of paramnesia are also common in general paralysis.

The special senses may in the earlier stages be exalted and hyperacute, but as the disease progresses they gradually become blunted and defective. Hallucinations of sight and hearing are present at some period in quite 50 per cent of the cases. Anæsthesia of the cutaneous nerves is almost invariable in the later stages. Analgesia is also a noticeable symptom. Ulnar analgesia (tested by pressure on the trunk of the ulnar nerve in the ulnar groove) is more frequent in general paralysis than in other forms of disease, even than in those which have other severe disturbances of sensibility. This sign (*ulnar sign*) is most common in tabetic forms of general paralysis. The absence of reflex action, when the finger is thrust between the fauces (*pharyngeal sign*), is also very commonly met with.

The eye symptoms are numerous and of great significance. Transitory defect of vision may

form an early symptom, later there may be optic neuritis, atrophy, and blindness. The *pupils* are generally unequal, often irregular in outline and defective in their reaction. Inequality, though common, is not pathognomonic of general paralysis. The reaction of the retino-iridal apparatus to stimuli is, however, of great importance. The rhythmic oscillations of the iris corresponding to the respiratory rhythm may be abolished; the normal dilatation of the pupil associated with bodily muscular movements may also be absent. Of more importance, however, is the failure to produce dilatation by acoustic, tactile, painful, and electrical stimuli. This "reflex dilatation" (Erb) may exist in spite of anæsthesia of the surface stimulated, and the failure of this reaction in cases of general paralysis is unaccounted for. It is difficult to obtain this reflex when the pupils are contracted. Sometimes, however, though no response can be obtained to stimulation of the skin, dilatation may result from emotional disturbance through the agency of the sympathetic. The so-called "*hippus*," or wide rhythmic oscillations occurring independently of respiration, is a symptom also seen in general paralysis. Consensual dilatation or contraction to light stimuli is sometimes impaired. Failure of one eye to react both to indirect and direct light stimuli indicates that eye to be the one affected, and the lesion to be a paralysing lesion affecting the sympathetic fibres of the defective side. When there is no marked contrast on shading both eyes, failure on one side to contract or dilate on direct light stimulation, failure on the same side to excite its fellow consensually, and its own consensual reaction, following excitation of the sound eye, then the lesion is probably in front of the chiasma, either retinal or affecting one optic nerve.

Bevan Lewis has indicated that in certain cases of general paralysis the initial contraction to concentrated light may be succeeded by a secondary dilatation—the pupil expanding widely in spite of the concentrated illumination, and he regards this as one of the earliest signs of a failing motor oculi nucleus—the centric expansions of the third nerve being readily exhausted.

There seems to be no definite relationship between pupillary inequality and the efficiency of the iridal reactions. Paralytic mydriasis is usually associated with exaggerated knee-jerks, and sometimes ankle clonus. Tongue tremors, marked affections of articulation, tremors of the lips and muscles of the lower facial group and of the hands. Tremors and twitching movements of the muscles of the brow are said to indicate alcohol as a probable factor of causation. Tabetic cases with iridoplegia have also altered reflexes. Romberg's sign may be present. Sometimes this sign cannot be obtained upon closing the eyes and approximating the feet, but if the body is bent forward swaying movements occur. Where the pupils are small or myotic the symptoms are usually tabetic. Accommodative reaction may be retained, but the Argyll-Robertson symptom is present. Usually the oculo-motor symptoms appear first—the loss of knee-jerk appearing later on.

Bevan Lewis tabulates the following five groups of symptoms as occurring in progressive paralysis of the insane:—

1. Paralytic mydriasis; a partial reflex iridoplegia (light). Increased myotatic irritability. Excessive facial tremor and speech troubles. Great optimism with profound dementia.

2. Mydriasis with associative iridoplegia rapidly passing into the cycloplegic form—an early symptom. Frequent myotatic excess, but no contractures. Late speech trouble. Acute excitement with frequent convulsions. Very rapidly fatal course. (Preponderance of syphilitic history.)

3. Spastic myosis; a complete reflex iridoplegia. Absent or greatly impaired knee-jerk. Failure of equilibration; locomotor ataxy, defective sensibility. Very defective articulation. Much optimism and excitement.

4. Late eye symptoms: paralytic mydriasis, a partial reflex iridoplegia (for light only). Ataxic paraplegia confined to lower extremities. (Arms do not participate.) Great facial ataxy with extreme troubles of speech. Epileptiform seizures ushering in pronounced mental enfeeblement.

5. No oculo-motor symptoms beyond occasional inequality. No contractures, but notable myotatic excess. No disturbance of equilibration, locomotion, or sensation. Speech troubles not pronounced. Epileptiform seizures very rare, but from the first progressive deepening dementia.

The handwriting is almost characteristic. Letters are often shaky, incomplete, detached, or illegible. In the early stages general paralytics also often write voluminously and quickly.

Seizures of various kinds occur either as early symptoms or as marked features of the later stages of the disease. They vary in severity from simple congestive to epileptiform or apoplectiform attacks. Some have merely slight attacks of giddiness, temporary loss of consciousness (*petit mal*), or transient loss of function of one of the special senses. In the stadium acutum congestive seizures are common and may resemble those of epilepsy or apoplexy. These attacks sometimes come on without warning, or they may be heralded by slight rise of temperature, confusion of ideas, or by some gastric disorder. They are irregular in their recurrence. Sometimes the epileptiform fits follow a definite line of development, and they usually leave behind them more physical and mental weakness than do ordinary epileptic fits. Apoplectiform seizures, on the other hand, are followed by less serious physical and mental troubles than in the case of true apoplexy. The

apoplectiform seizures may be attended with one-sided convulsions, with rigidity, convulsive movements, turning of the head to one side, and coma lasting for hours. They resemble apoplexies, except in that the condition is temporary, and any paralytic effects usually disappear in the course of a few days. Sometimes, however, mental dulness, drowsiness, or excitement follows the apoplectiform attacks, and may persist for days or weeks. Severe seizures may be followed by gradually increasing coma, palsy, and death. In fatal seizures associated with hemiplegia the brain may be affected by coarse disease or hæmorrhage. Tetaniform and hysteriform attacks also occur occasionally in general paralysis. The temperature is subject to irregular fluctuations quite independent of seizures. In maniacal states the temperature may rise slightly. Sometimes also in hypochondriacal forms there may be an evening rise. If excitement is protracted for several days there may be a rise, followed by a fall with the subsidence of the excitement. In some instances the temperature rises to 102° or 103° without any apparent physical cause. Subnormal temperatures are not common during the stadium acutum, but in the third stage they are relatively frequent. Fluctuations are relatively more frequent in patients with seizures, excitement, or with motor disorders. The circulation and pulse vary considerably, and in some cases indicating a primary cardiac enfeeblement. In maniacal cases with motor excitement the blood-pressure tends to become lowered: in quiet cases, on the other hand, it is raised. In the stadium acutum the pulse rate is relatively more frequent than is the case in the other stages.

The alimentary functions are usually active and well sustained throughout the course of the disease. During the early stages there may have been gastro-intestinal troubles, but in the stadium acutum the appetite usually becomes increased; organic sensations of discomfort seldom occur; sensations, which in the healthy usually indicate the condition of the stomach and intestines, are absent, and the want of knowledge as to whether they have had enough food leads to great increase in bodily weight. Progressive dysphagia is the rule, and is due to defective reflex action, incoördinate action, and later, paresis of the muscles of deglutition. Sometimes swallowing is almost impossible owing to bulbar lesions, or to pharyngeal and laryngeal anæsthesia. The dangers of death from choking are great, and careful attention to proper mincing of food is necessary.

The functions of the bladder and rectum become affected as the disease advances. The urine is passed involuntarily or retained, or there may be retention with incontinence due to an overfull bladder. The habits become foul, and the patient requires much personal attention. The sexual power, which may have been much exalted during earlier stages, now becomes impaired, and later lost.

The disorders of muscular and motor control are numerous. The lines of expression on the face become obliterated, and there is tremor of the muscles when movements are performed. This tremor is most marked about the muscles of the lips and tongue, and may be finely fibrillar or coarsely ataxic. The speech is difficult, hesitating, blurred, with slurring over labials, sibilants, or dentals. Words are jumbled together, or there is elision of syllables, or a species of stammering. These speech disorders may be due to defects of (1) *ideation*—either defective memory, or impaired association of ideas; defects of (2) *articulation*—ataxic or amnesic; or defects of (3) *phonation*—the vocal cords or the physical apparatus serving them being impaired.

Other symptoms are common, such as slavering, smacking the lips, sucking movements, grinding the teeth, or constant swallowing.

The gait may be ataxic, paretic, spastic, or the attempt to move may call forth trembling of the limbs. In some cases coarse trembling of the whole body occurs, when Romberg's test is attempted. Charcot's dictum that immobility is the finest movement of a soldier is here exemplified, and the co-ordination requisite for immobility is defective. Convulsive tremors without pyrexia, athetosis, and choreiform movements are sometimes seen.

The reflexes, superficial and deep, vary considerably. Exaggeration of knee-jerk is often associated with jerky, tremulous hand grasp, twitching of face, lips, tongue, and limbs, rigidity of proximal muscles of trunk and limbs, seizures, and dementia. Absence of knee-jerk is often associated with coarse ataxic gait, gross disturbances of sensation, cutaneous and articular, with consequent loss of so-called muscular sense, and megalomania.

Various vaso-motor symptoms occur, such as capillary congestions over the malar bones, erythematous and sudaminal affections of the skin, bed-sores, *tache cerebrale*, punctiform hæmorrhages under the skin after seizures, purpuroid blotches, bruise-like swellings, independently of any traumatic cause, othæmatomata, bullæ, eschars, unilateral local sweats, etc. Nutritive and trophic defects are evidenced by alterations in the body weight, dull, earthy, greasy, herpetic, ecthymatous, furfuraceous, or pigmented skin. Herpes zoster is common, and pemphigus parasyphiliticus occurs in the later stages of the stadium acutum. Furuncles and carbuncles appear. Bed-sores occur from pressure and excremental irritation, and assume a chronic type, or they may be acute and due to cerebral or spinal lesions, and appearing mostly after seizures.

The muscles sometimes undergo progressive

atrophy or fatty degeneration, or exhibit hæmatomata or extensive ecchymoses as the result of slight injury, etc. The bones tend to become more brittle and more easily fractured. Special arthropathies also sometimes occur.

Remissions of the symptoms may occur during the earlier stages of the disease. These remissions are most frequent in cases with maniacal excitement, and in which alcohol has been a factor in the causation. The remission may be complete, so that the patient may resume work for a time. Seldom is there more than one complete remission.

The *stadium dementiæ* is the third and final stage, and extends over a period of an average duration of one year. The dementia becomes more marked. There is loss of special and common sensation; objects, persons, and places are no longer recognised; the condition is almost purely negative, and the patient is incapable of self-care; occasionally there may be traces of former hallucinations or delusions, or brief attacks of excitement occur. With the mental hebetude there is neglect of the daily wants of nature; vesical and rectal troubles appear; ataxia gives way to paresis, and the patient becomes helpless and bed-ridden. The circulatory and respiratory functions are affected, and dyspnœa and syncope from heart failure are common. Tremors, spasms, atrophies, contractures, teeth-grinding, decubitus, and dysphagia become more pronounced, and death occurs through intestinal, renal, or vesical inflammations, bed-sores, septic infection, congestive seizures, or by œdema of lungs, pneumonia, embolism, or cerebral effusions. The duration of a typical case is therefore about three years. In galloping cases, however, death may ensue within a few months of the onset. In some tabetic cases the duration is of great length, and may extend over six or seven years. Some of the syphilitic and parasyphilitic cases also run through a protracted course.

The Diagnosis is often extremely difficult in the earlier stages. When we remember that the progressive paralysis may start with almost any form of mental or physical disorder the difficulty can be readily appreciated. Careful attention to any history of syphilis or alcohol in the case, and the presence of any of the physical symptoms already enumerated, will aid in coming to a decision. A differential diagnosis will have to be made from alcoholic disorders, which may simulate general paralysis in almost every particular, but from which the patient may recover; syphilitic cerebral affections, already considered; lead poisoning; convulsions of kidney disease; cerebral tumours; tabes; various systemic spinal lesions, with associated mental symptoms; disseminated sclerosis; paralysis agitans; sunstroke; epilepsy; apoplexy; various pseudo-general paralyses, which form sequelæ to fevers, malaria, traumatisms, etc.

Forms of General Paralysis.—The following clinical types are recognised:—(1) Acute or galloping general paralysis. The disease runs a very rapid course, and may terminate fatally in from two to three months from the onset. When the disease is ushered in by acute delirium there may be rise of temperature; physical and mental dissolution proceeds rapidly, and death ensues from exhaustion. Fits are not common. (2) Ordinary general paralysis, with mania and exaltation of ideas. Usually preceded by a period of depression, or hypochondriasis. Maniacal symptoms, with expansive benevolence and a feeling of well-being, supervene. The later stages show nothing characteristic to this group. (3) Melancholic, hypochondriacal, or stuperose general paralysis. Sometimes difficult to detect, but careful attention to the history of the case, syphilis as a factor of causation, and the presence of physical symptoms indicative of paralysis, render a diagnosis possible. Sometimes the depression passes off and mania supervenes. Remissions are not so common as in the maniacal forms. Fits of various kinds may occur. (4) General paralysis of the double form, or circular general paralysis. The first symptoms may be maniacal, followed by remission; next they may be melancholic and again followed by remission. These attacks may alternate and recur several times before a definite diagnosis can be arrived at; but sooner or later the physical symptoms show themselves and the disease progresses to a fatal end. (5) Progressive dementia with general paralysis. The mental symptoms are those of dementia from the outset. There is progressive enfeeblement of all the mental faculties. Fits are common. From being dull and heavy they become fat and fatuous, and in some cases without any maniacal or melancholic symptoms during the course of the disease. (6) Spinal general paralysis, that is, beginning with symptoms of systemic spinal disease. Tabes dorsalis, spastic paraplegia, or disseminated sclerosis may precede general paralysis by several years. Sometimes also general paralysis may be preceded by peripheral neuritis. (7) Developmental or adolescent general paralysis. Sometimes difficult to distinguish from disseminated sclerosis. Parental syphilis is almost invariable. These have already been referred to as cases of congenital parasyphilitic progressive paralysis. (8) Senile general paralysis. Often difficult to distinguish from senile dementia, with brain decay. The physical symptoms are seldom so characteristic of general paralysis as are the mental symptoms.

The physical signs are those most relied upon. The presence of excitement, exaltation, euphoria, and expansive benevolence, together with the characteristic affections of speech, fibrillary trembling of the muscles of the lips, tongue, face and limbs, spasmodic, convulsive, paralytic

seizures, alterations in the habits mental and physical, form a clinical picture sufficient to justify a diagnosis. It is seldom that the disease can be diagnosed from one symptom alone; in fact it is often only when the mental and physical signs form a definite clinical *tout ensemble* that the nature of the disease is evident. Much information can be obtained from the patient's friends as to alterations in his habits or modes of life. Before making a diagnosis the greatest care should be taken to ascertain whether alcohol has been a causal factor.

Prognosis.—The result of the disease is inevitable, but its course is variable. In some rare instances the disease becomes arrested, and the condition may remain stationary for many years. Undoubtedly general paralytics live longer in asylums than in their own homes. The relation of fits to the course of the disease is often interesting. Sometimes the seizures hasten the progress, or, on the other hand, they may cause an arrest or even remission. Some of the most rapid cases have had no seizures at all, and it is also noteworthy that fits of slight severity are sometimes associated with rapid progress of the disease. During the later months much will depend upon the amount of personal attention paid to the organic and nutritive functions.

Treatment.—Some cases of simple progressive dementia may be treated at home under suitable nursing and supervision. When there is excitement, marked depression, or certifiable delusions, however, it is advisable to send the patient to an asylum. Change of air or sea voyages are seldom beneficial. A simple life, with plenty of sleep and rest, moderate exercise, plain food, and attention to the bodily functions, are all that can be advised for many cases. Medicine and surgery can avail but little in staying the progress of the disease. For sleeplessness, paraldehyde in drachm doses proves of most service.

Generation, Female Organs of.

See also Acromegaly (*Symptoms, Generative Organs*); Embryology; Fallopian Tubes; Labour; Menstruation; Ovaries; Pregnancy; Puerperium; Teratology; Uterus; Vagina; Vulva.

The *Female Generative Apparatus* consists of the Internal Genitals, including the Uterus, Ovaries, Fallopian Tubes, and Parovarium, which lie within the Pelvis; the external Genitals, which are situated in the interfemoral space; and the Intermediate Organ, the Vagina, which connects together the Internal and the External Organs. The internal organs mainly rest on the inner aspect of the musculo-membranous diaphragm, which constitutes the pelvic floor; the external organs lie on its outer aspect; while the intermediate organ divides the floor into two segments, an anterior and a posterior. So closely are all these structures associated, that it is found to be convenient to consider the pelvis, osseous, ligamentous, muscular, and membranous, along with the organs of generation in the strict sense of the term.

The Osseous Pelvis.—(1) *Constituent Parts.*—The osseous pelvis is composed of four bones: a pair, the ossa innominata, and two single bones, the sacrum and coccyx. The innominate bones, with the assistance of the soft structures of the anterior abdominal wall, form the lateral and anterior walls of the pelvis; the sacrum and coccyx make up the posterior wall; in its osseous state the pelvis (or bason) is without a floor; this is supplied by the soft parts of the pelvic floor. Each of these four bones is composite, and was originally developed from several parts, which later on fused into one. Each os innominatum consists of three parts: an upper or iliac, an inferior or ischial, and an anterior or pubic, and these meet at the acetabulum, and become fused about the period of puberty; the sacrum consists of five segments, really five vertebræ, and these fuse a year or two later than the parts of the os innominatum; and the coccyx consists of four or five portions, really aborted vertebræ, which do not entirely fuse till late in reproductive life (thirty-five to forty years). About the same age, or later, the coccyx becomes firmly anchylosed to the sacrum, so that there is then one bone, sacrum-coccyx; up to this time the coccyx, which normally is directed forwards, can be pushed backwards, a movement known as retropulsion. The sacrum is jointed to the two innominate bones at the sacro-iliac joints or sychondroses, while the innominate bones meet by means of their pubic portions at the symphysis pubis. The whole pelvis is articulated to the lumbar part of the spine at the sacro-vertebral joint, where the upper part of the sacrum projects distinctly forwards in the promontory, while it is attached to the lower limbs at the acetabula or cotyloid cavities.

(2) *Osseous Pelvis as a whole.*—The pelvis as a whole has the form of an inverted truncated cone slightly flattened from before backwards; the base looks upwards and forwards, and the apex looks downwards and backwards. In its interior is a curvilinear, irregular canal, the upper part of which constitutes the large or false pelvis, and the lower the small or true pelvis. Its external surface may be generally described as roughened for the attachment of muscles, while its internal surface is smooth. The *false* pelvis may be more accurately defined as the part lying above the plane of the brim, bounded behind by the last two lumbar vertebræ, at the sides by the iliac bones, and anteriorly by the non-osseous abdominal wall. The plane of the brim in its turn may be defined as an imaginary surface, bounded behind by the sacro-vertebral promontory and the sacro-iliac synchrondoses, at the sides by the ilio-pectineal line, and in front by the upper border of the pubic bones and of the symphysis pubis. In the adult the normal non-pregnant genital organs do not lie in the cavity of the false pelvis; but in the fœtus and infant the upper part of the uterus and the annexa lie above the plane of the brim, and in pregnancy, after the fourth month, there is a similar but more marked ascent of the internal genitals. In the lateral walls are the internal iliac fossæ, which have a normal depth which it is difficult to express in figures, but which ought to be impressed upon the memory in order to distinguish it from the deepened state of the fossæ in the malacosteon deformed pelvis, and from the flattened out condition in the rachitic pelvis. There are two important diameters in the false pelvis: the interspinous, which joins together the two anterior superior iliac spines, and measures 24 cm.; and the intercristal, which passes between the widest apart portions of the iliac crests, and measures 27 cm. It is therefore convenient to remember that normally the intercristal diameter is slightly more than one inch longer than the interspinous. The intertrochanteric diameter is about 31 cm.

The *true* pelvis contains the generative organs, and requires a more detailed description than the false, for in it the phenomena of reproduction take place. It forms an irregular canal slightly enlarged at the middle, *i.e.* irregularly barrel-shaped; it is curved in the same manner as the anterior aspect of the sacrum, concave anteriorly. Its parts, diameters, inclination, planes, and axes call for description. It has three *parts*—brim, outlet, and cavity. The brim is the upper end of the canal, and is also known as the upper strait or pelvic isthmus, and has of course the boundaries of the plane of the brim (*vide supra*). In form it may be called oval or elliptical, but it is most correctly termed heart-shaped or cordiform, for the projection of the sacral promontory indents the oval posteriorly. The outlet of the true pelvis is also called the lower or perineal strait, and it is bounded behind by the tip of the coccyx, at the sides by the ischial tuberosities, and in front by the lower margin of the pubic bones and of the symphysis pubis. Its circumference is broken postero-laterally by the deep sacro-sciatic notches; these, however, are converted into foramina by the sacro-sciatic ligaments, and then the outlet has an ovate form, the broad end being posterior, and being indented by the projection forward of the tip of the coccyx. Anteriorly the circumference is broken by the pubic arch, which has a height of 5 cm. in the middle line, and a width varying from 3 cm. in the upper part to 9 cm. in the lower. Between the inlet and the outlet of the pelvis lies the cavity. It has a somewhat circular form. Its anterior wall has in the middle line a height of nearly 2 inches (4 to 5 cm.), and consists of the posterior aspect of the symphysis pubis and of the pubic and ischial bones as far back as a line dropped vertically downwards from the ilio-pectineal eminences. In this wall are the two obturator or thyroid foramina. The posterior wall is made up chiefly of the anterior curved surface of the sacrum, and has a height of about 6 inches (15 cm.) if the curve be followed. It is worth noting that on this wall, at the junction of the first and second segments of the sacrum, there is often a slight projection which might possibly be mistaken for the promontory which lies above it. The anterior boundaries of the posterior wall are lines dropped from the sacro-iliac synchondroses towards the insertion of the sacro-sciatic ligaments into the coccyx. Between the anterior and posterior walls lie the lateral walls, which have a height of about 4 inches (9 to 10 cm.). The anterior part of each lateral wall is called the anterior inclined plane; it looks backwards, inwards, and upwards, and corresponds to the internal aspect of the bone bearing the acetabulum, and of the body of the ischium and ischial tuberosity. The posterior part is called the posterior inclined plane; it looks forwards, inwards, and upwards, and corresponds to the internal aspect of the ischial spine, and of the sacro-sciatic ligaments and foramina. These inclined planes must on no account be confounded with the planes of the pelvis to be described below; the former have been supposed to play an important part in the mechanism of labour; the latter are geometrical conceptions useful in describing the mechanism of labour.

The *diameters* of the pelvis have a very considerable obstetric importance, and are described in relation to the brim, outlet, and cavity. The diameters at the brim are specially worthy of note; they are six in number—antero-posterior, transverse, two diagonals, sacro-cotyloidean, and sacro-pectineal. The antero-posterior is also called the sacro-pubic, sacro-suprapubic, or the

conjugate (d. conjugata vera); it passes from the middle of the promontory of the sacrum to the middle of the upper border of the symphysis pubis, and it measures from 11 to 11·5 cm. The transverse diameter at the brim passes between the widest apart portions of the ilio-pectineal lines, and measures 13·5 cm. The diagonal or oblique diameters of the brim are two in number, right and left; the former passes from the right sacro-iliac synchondrosis behind to the left pectineal eminence, while the left passes from the left sacro-iliac behind to the right pectineal eminence in front; each measures 12·5 cm., but strictly speaking, and on account of the slight physiological deviation of the pelvis to the left side, the right oblique is slightly longer than the left. The oblique diameters, it will also be noted, connect together four bony landmarks, which have been called the cardinal points of Capuron, and so divide the brim into quadrants. The sacro-cotyloidean diameter is sometimes referred to; it passes from the middle of the sacral promontory to the point on the inside of the pelvis which corresponds to the upper and posterior part of the cotyloid cavity on the outside; there is therefore one on each side, right and left, and it measures 9 cm. The sacro-pectineal or diameter of Burns passes from the middle of the promontory to the crest of the pubes immediately above the obturator foramen; it has a length of 10 cm. It is useful to remember that the shortest antero-posterior diameter of the pelvis is not the conjugata vera, but one drawn from the middle of the promontory to the nearest point on the posterior surface of the symphysis pubis, a point which is usually about 5 mm. below the upper margin; this is called the diameter minima; and its length is from 10·5 to 11 cm. The pelvic diameters at the outlet are four in number: antero-posterior, transverse, and obliques. The antero-posterior passes from the tip of the coccyx to the lower border of the symphysis pubis, and measures with the coccyx pressed backwards, 12 cm.; with it in its usual position, 10 cm. The transverse diameter is the distance between the tubera ischii; it measures 11 cm. The obliques are difficult of exact definition, and they have a varying length in parturition, as the sacro-sciatic ligaments yield a little to the fœtal head passing through the outlet. In the cavity of the pelvis various diameters may be measured, but the following are those of most interest. There is the antero-posterior drawn from the third sacral vertebra to the middle of the posterior surface of the symphysis, and measuring 12·5 cm.; there is the transverse, passing between the areas of bone on the inside of the pelvis corresponding to the acetabula on the outside, measuring 12 cm.; there is another transverse diameter which is sometimes of importance—the bi-ischial, the distance between the ischial spines, measuring 10 cm.; and, finally, there are the obliques of the cavity, right and left, passing from the sciatic foramen on one side to the obturator foramen on the other, and measuring about 13·5 cm. (Fig. 1).

In addition to the diameters which have been described there are two others called conjugates, which have a very considerable clinical importance, for they can be measured during life. These are the diagonal conjugate or sacro-subpubic diameter, and the external conjugate or diameter of Baudelocque. The former is drawn from the middle of the promontory to the lower border of the symphysis pubis, and measures from 12 to 12·5 cm.; from it the conjugata vera can be deduced by the subtraction of 2 cm. The external conjugate is measured from the spine of the last lumbar vertebra to the upper border of the symphysis pubis, and in order to get the right spinous process in the living subject it is well to draw a line connecting the depressions representing the posterior superior iliac spines and the second spinous process, above this is that of the fifth lumbar vertebra. This diameter is 18 cm. in the osseous pelvis, and 19·5 cm. in the living woman.

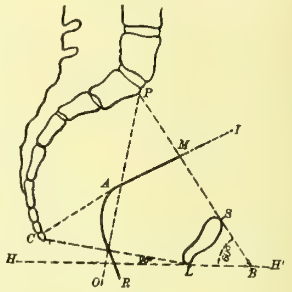


Fig. 1.—Axes of the pelvis: *P*, promontory of the sacrum; *C*, coccyx; *S*, symphysis pubis; *I*, umbilicus; *PS*, plane of the superior strait; *CL*, plane of the inferior strait; *HH'*, line of the horizon; *IMA*, axis of the superior strait; *AR*, axis of inferior strait; *MAR*, curve of Carus; *PBL*, obliquity of superior strait; *CLH*, obliquity of the inferior strait. (After Dorland.)

By the *inclination* or *obliquity* of the pelvis is meant that it is not in the axis of the body, but placed obliquely to it, as is shown by the fact that in the erect posture the sacral promontory is 9 cm. above the upper border of the symphysis pubis, while the tip of the coccyx is about 2 cm. above the level of the lower margin of the symphysis. The degree of the inclination of the inlet is found by measuring the angle which the antero-posterior diameter of the brim when prolonged makes with the horizon; it is about 60°; in the same way the inclination of the outlet is 10° or 11°, but when the coccyx is pressed backwards the antero-posterior diameter corresponds with the horizon. In the recumbent posture with the sacrum raised the pelvic inclination is 25°; in the same posture with the legs hanging freely over the edge of the table the greatest inclination is obtained (about 10°).

Planes of the pelvis are often referred to in descriptions of the mechanism of labour. The plane of any part of the pelvis is a surface supposed to touch all points in the circumference of that part; thus the plane of the brim is an

imaginary surface touching all points of the circumference of the inlet. It is thus possible to draw a large number of such planes in the pelvis, and the form of each is represented by the relative length of the various diameters thereof. The plane of the inlet has an inclination of 60°, that of the outlet is nearly horizontal. It is noteworthy that the plane with the smallest diameter, that between the ischial spines, is not at the outlet but just above it; it is that between

FIG. 2.—Male pelvis seen from the front.

the apex of the sacrum and the lower border of the symphysis; its antero-posterior diameter also does not vary, for the sacrum cannot be pressed backwards as can the coccyx. The *axes* of the pelvis are lines drawn perpendicularly to the centre of the planes, and there are in consequence as many axes as there are planes. The axis of the inlet is directed upwards and forwards, and corresponds with a line passing through the umbilicus and the lower end of the coccyx. The

FIG. 3.—Female pelvis seen from the front.

axes of the outlet is directed downwards and slightly backwards. If the axes of the brim, outlet, and cavity of the pelvis be joined together, the result will be a curved line, and this is called the central line, the guiding line, or the line of direction of the pelvis; it indicates the curve which the fœtal head follows in its descent through the pelvic canal in labour (Fig. 1).

Pelvic Differences due to Sex, Age, and Race.—The sexual differences in the osseous pelvis are early impressed upon it, as Fehling pointed out some years ago, and as Arthur Thomson has recently shown. According to the latter (*Journ. of Anat. and Physiol.* xxxiii. 359, 1899) the essential sexual differences are as well defined during fœtal life as they are in the adult. These are briefly as follow:—In the female the walls are thinner and their bony prominences are less marked; the vertical dimensions are less, the transverse dimensions are greater, and so is the antero-posterior diameter in the cavity; the acetabular cavities are farther apart, and the false pelvis is wider, while the true pelvis is more rounded; the pelvic inclination is greater; the promontory is less projecting, and the sacrum is broader and shorter; the obturator foramina are larger, and triangular rather than oval; the pubic arch is broader and more rounded in its upper part, the angle being 90° to 100° instead of 70° or 80°; the depth of the symphysis is less; and, finally, the sacro-coccygeal joint is more movable than in the male. In order to contrast the age differences of the pelvis the

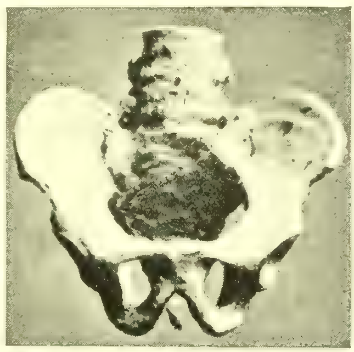


FIG. 4.—Pelvis of seven months' fœtus. (After A. Thomson.)

fœtal may be compared with the adult female. In the former the promontory is high, the pelvic inclination being 75° to 80°, and the sacrum and coccyx are straight; in the latter the promontory is not so high, it projects forward more, the pelvic inclination is from 55° to 60°, and the sacrum and coccyx are concave; in the fœtus all the diameters are relatively smaller in the true pelvis than in the adult, and the transverse at the brim is only slightly longer than the conjugate instead of being markedly so as in the adult; it is usually stated that the sacrum in the fœtus is relatively narrower than in adult life, but Arthur Thomson (*loc. cit.*) denies this; and, finally, the ischial tuberosities are nearer to each other at the outlet in the fœtal pelvis than are the ischial spines, while in the adult the spines are nearer than the tuberosities. The racial differences of the pelvis have not yet been fully worked out; but it would seem that among the primitive races of America and Australia, and in the case of the Malays, the diameters at the pelvic brim approximate more closely to each other, so that the antero-posterior and the transverse come to have nearly the same measurement. In other words, the inferior races

have a pelvis with a more rounded inlet. In addition to the sexual, age, and racial differences which have been described, it must be remembered that pelves of the same age, sex, and race may nevertheless show peculiarities which cannot be called pathological, and must be regarded as individual; no two pelves probably are exactly alike. Further, it is generally found that the pelvis is not symmetrical, but shows sinistro-scoliosis, the symphysis being drawn towards the left (Figs. 2, 3, 4).

The Ligamentous Pelvis.—The four bones which make up the osseous pelvis are articulated to each other at the two sacro-iliac synchondroses, at the sacro-coccygeal joint, and at the symphysis pubis. These joints are amphiarthroses; but the symphysis pubis has by some been considered as a mixed joint, arthrodial behind and amphi-arthrodial in front, while others have looked upon the sacro-coccygeal as entirely arthrodial. The sacro-iliac articulations, consisting of the auricular surfaces of the sacrum and innominate bones, are held together by four ligaments (anterior, superior, inferior, and posterior); but the great and small sacro-sciatic ligaments which pass from the sacrum and coccyx to the ischial bones also play a part in maintaining the apposition of the articular surfaces. The synovial membrane is small in extent; and the amount of movement possible at the sacro-iliac joints is very limited, but is slightly increased in pregnancy, and is also greater if the symphysis pubis be divided. The symphysis pubis has considerable importance at the present time through the revival of the operation of symphysiotomy. The articular surfaces are oval, and between them is an interosseous fibro-cartilage; they are held together by anterior, posterior, superior, and inferior, or subpubic ligaments. A slight degree of movement is possible at this joint, which is increased if the pubic bones be divided through the obturator foramina; on account of slight swelling and increased moisture of the tissues there is also more marked mobility in pregnancy. An interosseous fibro-cartilage and peripheral ligaments are met with in connection with the sacro-coccygeal joint, which has a considerable range of mobility up to mid-life; but it has to be borne in mind that some movement also occurs between the segments of the coccyx which are united by rudimentary amphiarthroses. Indeed, some writers are of opinion that the movement backwards of the coccyx which occurs in labour really takes place at the first inter-coccygeal joint. The pelvis as a whole is attached to the lumbar part of the spinal column by a median symphysis or amphiarthrosis and by two lateral arthrodia; it is articulated to the lower limbs at the hip-joints; but these joints do not require special description here. The other ligamentous structures of the pelvis are the obturator membranes and Poupart's ligaments: the former close in the obturator foramina and complete the anterior pelvic wall; the latter stretch from the anterior-superior iliac spine to the spine of the pubic bone on each side, and the part of it which is attached to the pubic crest is called the ligament of Gimbernat.

Internal Generative Organs.—The internal female genital organs include the ovaries, the Fallopian tubes, and the uterus; and along with these may be described the parovarium. The organs will first be considered separately, and later their relation to the soft structures filling up the pelvic outlet will be taken up.

Ovaries.—The ovaries are two small bodies of an ovoid form lying one on each side of the uterus and having the function of forming ova. They are the distinctive organs of the female. They are situated at the level of the pelvic brim and are attached to the posterior surface of the broad ligament of the uterus by the anterior border alone, and not, as has sometimes been stated, by the anterior surface. Their exact position and the direction of their long axis vary slightly with the empty or full condition of the neighbouring hollow viscera. Since they have an ovoid or almond shape they possess two surfaces, two ends, and two borders or margins; and the long axis is directed obliquely outwards and upwards towards the side of the pelvic brim. The anterior margin (hilum) is attached to the broad ligament, while the posterior is free; the surfaces are better described as outer and inner than anterior and posterior, and of these the latter is the more convex, the former being flattened; the lower and inner end (uterine pole) is the smaller, and is united to the uterus by the ovarian ligament; the outer end (tubal pole) is more rounded, and is attached to the fimbriated extremity of the Fallopian tube by the pen-shaped ovarian fimbria. Each ovary has dimensions which may roughly be expressed as length 3 cm., width 2 cm., and thickness 1 cm., or in inches $1\frac{1}{3}$ by $\frac{3}{4}$ by $\frac{3}{8}$; but the size varies much with (*a*) age, (*b*) menstruation, and (*c*) pregnancy, the maximum being attained about six weeks after parturition. The weight varies from 90 to 135 grains, falling as low as 20 grains in old age. The Fallopian tube lies above the ovary in the broad ligament, and its outer end encircles the outer end of the ovary; between the two and lying in the broad ligament is the parovarium. The ovary itself does not lie between the layers of the broad ligament, but is simply attached to the posterior surface of it by the hilum, at which point enter the vessels and nerves; here can be seen a white line (Farre-Waldeyer's line).

The structure of the ovary is somewhat complicated. To the naked eye the gland has a pearly or even pinkish hue, with dusky blue spots here and there in it, which represent follicles containing ova, and it shows also yellow spots or old and ruptured follicles. In early life the surface is

smooth, but later it becomes irregular. Two zones can be distinguished, a vascular zone or medulla or paroöphoron, and a parenchymatous zone or cortex or oöphoron. The former is softer in consistence, pink in colour, and contains large vessels, which enter from the broad ligament; the latter is firmer, white, and less vascular, and is dotted with small pits (follicles) (Fig. 5).

With the microscope it can be made out that the zona vasculosa is made up of loose connective tissue with some unstriped muscular fibres, large tortuous blood-vessels, nerves, and lymphatics, and some isolated tubular relics of the mesonephros. The parenchymatous zone shows most externally a layer of germinal columnar epithelial cells, which stops at the hilum at the white line, becoming continuous with the endothelium of

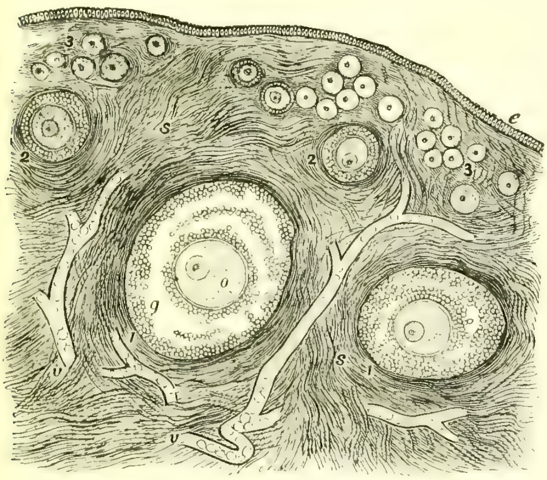


Fig. 5.—Section of ovary, showing large and small ovi-sacs, epithelial covering, and vessels. (After Baldy.)

the peritoneum of the broad ligament. Beneath this is in some ovaries (especially from old women) a connective tissue layer, the tunica albuginea. The greater part of the parenchymatous zone, however, is composed of the folliculiferous stroma. In it are found the Graafian follicles, from 36,000 to 40,000 in number in each ovary in the new-born infant, along with connective tissue, vessels, and nerves. The follicles are of three kinds, primary, growing, and ripe or mature: the primary and growing follicles are smaller than the ripe ones and lie nearer the surface, although occasionally a ripe one is found at the periphery. In the primary follicles are the primitive ova with a few somewhat flattened epithelial cells to form a covering; in the growing follicles the epithelial cells have become more cubical in shape, and form several layers round the ovum, which shows the commencement of a zona pellucida. The ripe follicle is a complicated structure: from without inwards can be recognised (*a*) a fibrous or external membrane or tunic, consisting of spindle-shaped, round, and stellate cells; (*b*) a membrana propria or internal tunic, consisting of a delicate layer of connective tissue; (*c*) a membrana granulosa or stratum granulosum, consisting of two or more layers of nucleated columnar cells, lining the follicle and massed together at one point into a clump of cells, forming the discus proligerus or cumulus oöphorus, and containing the ovum itself; (*d*) the liquor folliculi, a clear, yellow fluid, containing much paralbumin; and (*e*) the ovum, composed from without inwards of (1) the ovular epithelium, consisting of the granular epithelial cells of the membrana granulosa, which lie nearest to the ovum, and to which the name corona radiata is sometimes given; (2) the zona pellucida or vitelline membrane, which also shows a striation; (3) the perivitelline space; (4) the protoplasmic portion of the vitellus, consisting of an outer clear part and an inner more granular portion; (5) the deutoplasmic part of the vitellus; and (6) the germinal vesicle or nucleus, situated excentrically, and in it the germinal spot or nucleolus which has shown, according to some observers, amœboid movements. A ripe Graafian follicle, such as has been described, is ready to play its part in reproduction: in ovulation the follicle enlarges, passes from the centre towards the periphery of the ovary, during which time the ovum is throwing off two small rounded bodies, the polar globules, and the nucleus is undergoing karyokinetic changes; then the follicle ruptures, the ovum and part of its surrounding cells of the membrana granulosa escaping to be caught in the outer end of the Fallopian tube and carried along the tube into the uterus. The changes which occur in the follicle after rupture lead to the formation of the structure known as the corpus luteum. This structure is composed of blood-clot and of an ingrowth of the vascular wall of the follicle into the cavity from which the ovum has escaped; gradually the wall becomes thrown into folds and less room is available for the clot, which soon occupies only the centre of the corpus; the corrugated wall is first of a bright and later of a pale yellow colour; and finally the position of the corpus luteum is represented by a white cicatrix (corpus albicans). The corpus luteum which follows an ovulation unaccompanied by pregnancy runs through all the above described stages much more quickly than one which ensues upon impregnation; otherwise there is no essential difference between the corpus luteum of pregnancy or corpus verum and the corpus luteum of menstruation (so-called). The colour is due to the presence of lutein.

The arterial supply of the ovaries is from the ovarians which come from the aorta; the veins pass to the pampiniform plexus in the broad ligaments and thence to the ovarian veins, of which one, the right, opens into the vena cava inferior and has a valve, while the other, the left, joins the renal vein at right angles, and

has no valve. The lymphatics pass to the lumbar glands, and the nerves come from the inferior hypogastric plexus of the sympathetic and from the sacral nerves.

In the fœtus and young infant the ovaries lie above the plane of the brim of the pelvis, have an almost cylindrical elongated form, and show on section a much greater number of follicles than is to be observed in later life. The glands are chiefly derived from the germinal epithelium of the genital ridge; but a small part is developed from the mesonephros.

Fallopian Tubes.—The oviducts or Fallopian tubes are two sinuous tubes which occupy the upper part of the middle fold of the broad ligament, called the mesosalpinx or the mesentery of the tube. At the inner end they arise from the upper angle of the uterus, while the outer extremity opens into the general peritoneal cavity, but is connected with the ovary by the ovarian fimbria, and with the pelvic brim by means of the free fold of the broad ligament or infundibulo-pelvic ligament. Each tube is shaped like a shepherd's crook; the first part nearest to the uterus runs out straight, the second part curves straight outwards and forwards, and the third backwards and inwards. The first part, or isthmus, is the narrowest, and through it the uterine and tubal cavities are continuous; the second, or ampulla, is much wider; while the third, or infundibulum or fimbriated extremity, is expanded like the mouth of a trumpet and consists of the abdominal opening (ostium abdominale) and of the fimbriæ or fringes surrounding it. The right tube is usually a little longer than the left, the measurements being 11 cm. for the former and 10 cm. for the latter. The diameter near the inner end is about 4 mm., farther out it measures from 7 to 9 mm., and the ostium abdominale has a diameter of from 2 to 5 mm. A neck of constriction just internal to the infundibulum has been described; this may indicate an external sphincter of the tube. The fimbriæ are primary and secondary, or major and minor; the former are three or five in number; and in the intervals between them are the secondary, which are more numerous. The fimbriæ give to the end of the tube the appearance which has been termed "morsus diaboli" (Fig. 6).

The structure of the tubes is like that of other hollow viscera. To the naked eye three coats or layers are recognisable: an external or serous formed of the peritoneum of the mesosalpinx; a middle or muscular made up of an outer longitudinal layer of fibres and an inner circular; and an internal or mucous thrown into folds, which are continuous with the fimbriæ at the ostium abdominale. In the isthmus the tubal folds or rugæ are simple, and are arranged longitudinally, and the section of the tube is stellate; in the ampulla their arrangement is much more complicated, there are from three to five primary folds, and from eight to ten secondary ones, and all these branch, so that on section the tube has a dendritic appearance; and at the infundibular end the folds become continuous with the fimbriæ, primary folds with primary fimbriæ, and secondary with secondary. Under the microscope the outer coat has the ordinary appearance of a serous membrane; but it is noteworthy that at the ostium abdominale the flat cells of the peritoneum stop abruptly and give place to the ciliated columnar cells of the mucous membrane at the muco-peritoneal line, constituting a unique occurrence in the histology of the body. With regard to the muscular coat it is to be observed that there is a marked increase in the number of the circular fibres at the point where the tube passes through the uterine wall and also at the neck of constriction (above referred to) just internal to the infundibulum. The microscope has shown that the outer longitudinal and inner circular arrangement of the musculature is not constant, for in some specimens longitudinal fibres are found lying internal to the circular and bulging into the bases of the folds of the mucosa. A submucous layer is not invariably present, but is occasionally found, especially in young women, as a thin layer most evident in the isthmus. It consists of round and spindle-shaped cells of embryonic character. The folds of the mucosa have a basis of similar cells with connective tissue fibres, a central artery, and are covered by columnar ciliated epithelium. The appearances of the tube in transverse section are distinctly suggestive of the presence of glands, but some

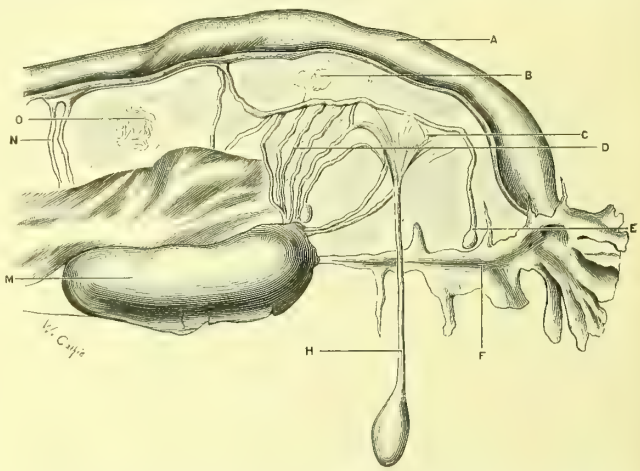


Fig. 6.—Fallopian tube, parovarium, and ovary (seen from the front). A, Fallopian tube; B, relic of mesonephros; C, curved tubules of parovarium; D, vertical tubules; E, cyst, of Gartner's duct; F, pen-shaped ovarian fimbria; H, pedunculated cyst of parovarium; M, left ovary; N and O, tubular relics of mesonephros. (After Ballantyne and Williams.)

authors deny their existence. To the outer end of the tube or to its fimbriæ there is sometimes found attached a stalked cyst; this is the true hydatid of Morgagni; it is not to be confounded with the pedunculated cysts growing from the parovarium. In the fœtus the tubes present several spiral convolutions.

Organ of Rosenmüller, or Parovarium.—Between the folds of the mesosalpinx there lies, in addition to the Fallopian tube, the structure known as the organ of Rosenmüller, or Parovarium. It consists of mesonephric tubular relics, and has also been termed the corpus pampiniforme or corpus conicum, or the epoöphoron. The organ is trapezoid, not triangular in form, and measures at its base from 1·5 to 3·5 cm. transversely, while at its narrow end near the ovary its breadth varies from 4 mm. to 3 cm.; its height is from 1·3 to 3 cm. It lies below the Fallopian tube and above the ovary, and is not free between the folds of the broad ligament, but is attached to the anterior one. It consists of a horizontal duct, the remnant of the upper third of the Wolffian duct, and of a number of tubules running more or less vertically downwards from it. The inner vertical tubules pass towards the hilum of the ovary, some reaching it, others falling short of it; the outer ones have a more curved course, and do not reach the ovary at all, but end in flask-like dilatations. The vertical tubules disappear near the hilum of the ovary in an obscure area (locus obscurior of Rosenmüller). In this place, as shown by the microscope, the tubules in an atrophied condition form a network, the rete ovarii, and some traverse the substance of the paroöphoron. The curved tubules most commonly lie in a free fold of the anterior layer of the mesosalpinx, and they often have attached to them cysts which have been called hydatids of Morgagni, but which really differ from that structure, as has been already stated. The horizontal duct shows a lumen, and has a wall composed of fibrous or connective tissue with some non-striped muscular fibres, and is lined with columnar non-ciliated epithelium. Few of the vertical tubules exhibit anything like a lumen; they have a thin covering of connective tissue, and are lined internally by columnar epithelium near their upper end, and by cubical cells nearer the ovary. Isolated tubular structures not included in the organ of Rosenmüller are sometimes found between that body and the uterus. These are also relics of the sexual part of the mesonephros, and probably correspond to the vas aberrans of Haller in the male. Granular areas may also be found at various spots between the layers of the mesosalpinx, some lying between the organ of Rosenmüller and the uterus, and others between the base of that organ and the Fallopian tube. In the fœtus and child they may be seen between the tubules of the parovarium itself. Sessile cysts sometimes take their origin in them. They consist of mixed tissue, partly fibro-cellular and partly fatty, with a trace of fine canaliculi with a choked lumen. Ballantyne and Williams think that they represent relics of the urinary part of the mesonephros, and that they correspond in the male to the organ of Giraldès or paradidymis of Waldeyer (Fig. 6).

Uterus.—The uterus is a hollow, thick-walled organ lying in the middle of the pelvis between the rectum behind and the bladder in front. From it the menstrual discharge comes, and in it the phenomena of the development and growth of the fœtus take place. The question of its position, once much discussed, may now be regarded as settled. It is to a certain extent a movable organ, but its normal position, as determined both by clinical examination and by the study of frozen sections, may be defined as one of anteversion with a slight degree of anteflexion; it has a curve something like that of the anterior surface of the sacrum, and rests upon the posterior surface of the bladder; its lower end or cervix is directed downwards and backwards, and the whole organ is slightly twisted, so that its right upper angle is a little anterior to the left one. In shape it is a cone flattened from before backwards, with its base or fundus above, and its apex or cervix below. Below its middle third there is a slight constriction, the isthmus, which divides it into a body or corpus and a neck or cervix. The lower end of the cervix shows an opening, the os externum uteri; while the upper part of the body is the fundus, which is slightly arched, and to the angles of which are attached the Fallopian tubes. Its size varies with age, with certain physiological states, such as pregnancy, and of course with pathological conditions; but it may be stated generally that in the adult virgin it has a length of 7·5 cm. (3 inches), a breadth of 4 cm. (about 2 inches), and a thickness of 2·5 cm. (1 inch). In a woman who has borne children all these measurements are slightly increased. The length of the cavity from os externum to fundus is 6 cm. or $2\frac{1}{2}$ inches. The thickness of the wall is 1 cm. Its weight is from 40 to 50 grammes. Approximately three-fifths of the whole length belong to the body of the uterus, and two-fifths to the cervix. The body has a form which is somewhat triangular in the virgin, and tends to the globular in a woman who has borne children; it has an anterior and a posterior surface, of which the former is flatter, two lateral borders to which the broad ligaments are attached, a superior border or fundus uteri which is free, two upper angles from which the inner ends of the tubes arise, and a lower end continuous with the cervix at the isthmus. A coronal section of the body shows the triangular shape of the cavity, while a sagittal section demonstrates that the anterior and posterior walls are in contact, the

cavity being therefore a virtual one. The cervix is nearly cylindrical in form (it is slightly fusiform), and projects into the upper end of the vagina; its cavity is fusiform. It communicates with the cavity of the body at the isthmus by means of the os uteri internum, and with the vaginal canal by the os uteri externum. It has sometimes been divided into two parts, a supra-vaginal and a vaginal (portio vaginalis); but as it does not project equally into the upper end of the vagina, it is more correct to regard it as consisting of three portions, a supra-vaginal, an intermediate, and an intra-vaginal portion. The last named is the unattached part of the cervix; the intermediate is attached to the bladder in front, but is free behind; while the supra-vaginal is attached to the vaginal roof behind and to the bladder in front. The os uteri internum is a circular opening; the os uteri externum, or os tincæ, is a narrow, transversely situated orifice in the vaginal portion which divides this part of the cervix into two lips, anterior and posterior. It is noteworthy that the occurrence of labour shows that the uterus, which in the unimpregnated condition consists evidently of the two portions described above as body and cervix, really is made up of three parts, body, lower uterine segment, and cervix. Just before the opening up of the os uteri internum there can be recognised in the pregnant uterus the body or upper uterine segment with thick, contracted walls, the distended lower uterine segment, and the cervical canal. By some the lower segment is regarded as belonging to the body, and by others as due to the opening up of the upper part of the cervical canal. The former is the more probable view. The relations of the uterus to the pelvic floor will be considered later (Fig. 7).

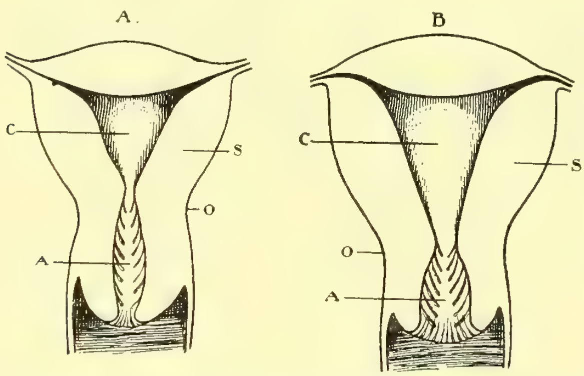


Fig. 7.—Coronal section of a nulliparous (A) and of a multiparous (B) uterus: A, cavity of the cervix and arbor vitæ; C, cavity of the body; O, constriction between body and cervix, the os uteri internum; S, wall of body. (After Tarnier.)

Structure of the Uterus.—With the naked eye it can be recognised that there are three coats or tunics—the external, serous coat, or perimetrium; the middle muscular coat, or mesometrium; and the internal mucous coat, or endometrium. The serous coat is formed by the general peritoneal lining of the pelvis. It may be noted that it covers the anterior surface of the uterus as low as the level of the os internum uteri, where it passes up on to the posterior surface of the bladder to form the utero-vesical pouch of peritoneum. Posteriorly it covers the posterior wall of the uterus, and descends as the pouch of Douglas behind the upper part of the vaginal roof as low as the level of the os externum uteri. From this point it ascends again on to the anterior rectal wall. Laterally the anterior and posterior layers of peritoneum meet at the borders of the uterus to form the broad ligaments of the organ. At the fundus the peritoneal coat passes directly from the anterior to the posterior surface. In the lower animals the musculature of the uterus is clearly divisible into two layers, an internal circular and an outer longitudinal, the former being a strongly developed stratum, and the latter a feebly marked one; but in the human uterus there is no such distinct arrangement. With the supervention of pregnancy, however, a notable hypertrophy of the muscular coat of the uterus occurs, and it is then possible to recognise three layers; but even then the distinction between them is never so marked as in the uteri of the lower animals. There is an inner layer immediately beneath the mucous membrane in which the fibres are arranged chiefly in longitudinal bundles. This has by some been regarded as a hypertrophied muscularis mucosæ. The middle layer is much the thickest, and while it is largely composed of fibres running in a circular direction, it also contains not a few which pass longitudinally, and from the peritoneal surface towards the mucous. In this way a thick felt-work is formed in the meshes of which lie the large blood-vessels of the uterine wall. The external layer is made up of longitudinal bundles with some obliquely and circularly disposed ones interspersed. Fibres from it pass into the broad ligaments, and also into the utero-sacral bands which run backwards to the sacrum below the peritoneal lining of the pouch of Douglas, dividing that pouch into three compartments, a central and a right and left lateral. The musculature of the cervix is largely continuous with the middle layer of the body. The mucous membrane is a thick coat, and has a smooth surface in the body of the organ, while in the cervical canal it is thrown into ridges, a vertical one with lateral ones slanting upwards and outwards from it, and to this arrangement the fanciful name of the arbor vitæ has been given. With the help of the microscope it can be made out that the serous coat has the ordinary histological features of the peritoneum, viz. a basis of delicate fibrous and connective

tissue supporting large endothelial cells, while the muscular layer is found to be composed of very large unstriped muscle fibres, sometimes branched and with elongated nuclei, in a matrix of fine connective tissue and elastic fibres. In the muscular coat are also many arteries with thick walls and a convoluted intima, and large thin-walled veins with no valves. The microscopic characters of the mucous membrane of the body differ from those of the cervix, and require a separate description. Further, the appearances are much altered by such physiological conditions as menstruation and pregnancy. The following description refers to the mucous membrane of the resting (non-menstruating) virgin uterus:—

It measures 1 mm. in thickness. It is made up of a loose plexus of embryonic connective tissue cells branching and anastomosing with here and there more elongated and fusiform cells with oval nuclei; in the interstices are many leucocytes, and in its general appearance it suggests the structure of lymphoid organs. On the uterine surface is a single layer of ciliated columnar epithelial cells. On the surface also open the mouths of the uterine glands; these are tubular and extend down through the stroma, and often bifurcate at their lower ends, some of which reach and some even penetrate the muscular coat. Their direction is first perpendicular, and then oblique to the surface. They are lined by ciliated columnar epithelium upon a nucleated basement membrane, but the latter structure has not been recognised by all. Further, some observers regard these structures as not truly glandular in nature, but as simple pits or diverticula of epithelium, the mucous membrane being really a lymphatic tissue. In support of this view Leopold states that the connective tissue bundles are surrounded by endothelium, and therefore constitute lymph spaces. In the cervical canal the mucosa is thicker than in the body of the uterus; it is firmer in consistence, and its lining columnar epithelium is ciliated only on the tops of the ridges of the arbor vitæ. It possesses numerous racemose glands lined with cubical epithelium. At the level of the os externum uteri there is usually a sharp transition between the columnar epithelium of the cervical canal and the squamous epithelium reflected on to the outside of the vaginal portion of the cervix from the vaginal walls; but it is noteworthy that in the infant at birth there is often no marked line of separation between the two kinds of epithelium, the columnar lining of the cervical canal extending for a limited distance over the vaginal aspect of the cervix, and giving rise to the condition which has been called congenital cervical erosion. The so-called ovula Nabothi are retention cysts of the cervical glands. During menstruation congestion of the mucous membrane of the body of the uterus occurs, accompanied by an exfoliation of a greater or smaller part of it; during pregnancy it gives attachment to the fertilised ovum, and forms the maternal part of the placenta.

The arterial blood-supply of the uterus is derived from two sources: the greater part comes from the uterine artery, a branch of the anterior division of the internal iliac, which passes in the broad ligament to a point near the os externum, where it turns upwards along the lateral border of the uterus, giving off transverse branches (the curling arteries) to that organ on the way, and one of these is large, is given off near the os internum, and uniting with that of the opposite side, forms the circular artery; a small part comes from the ovarian artery, whose terminal branches anastomose with those of the uterine. It is well to remember that the uterine artery which at a point opposite the os externum lies below and in front of the ureter then crosses over it and comes to lie behind its level. The veins of the uterus form a very rich network below the serous coat, and communicate with the vaginal and vesical veins, and also with the pampiniform plexus; they open into the internal iliac vein directly or into the ovarian vein. The lymphatics join those from the tube and ovary, and pass between the layers of the broad ligament to the lumbar glands; those from the cervix and upper part of the vagina pass together to the hypogastric and obturator glands. The nervous supply is derived in part from the hypogastric plexus of the sympathetic, and in part from the second, third, and fourth sacral spinal nerves; on each side of the uterus at the level of the isthmus these nerves unite in forming a large plexus sometimes called the cervical ganglion.

The uterus and vagina are developed from the fused portion of the two Müllerian ducts. The upper part of each duct remains separate as the Fallopian tube. In the new-born infant the uterus is partly an abdominal organ, for a portion varying from a half to a third lies above the plane of the brim of the pelvis; the cervix is relatively much larger both in length and thickness than the body of the organ; and the folds of the arbor vitæ are prolonged in the interior quite to the fundus.

Ligaments of the Uterus.—The uterus partly is embedded in and partly rests upon the pelvic floor, but it is also to some extent supported by the various ligamentous structures which sling it in the pelvic cavity. These are the round ligaments, the broad ligaments, the utero-sacral and the utero-vesical ligaments. The round ligaments are two fibro-muscular structures 13 cm. in length, which arise from the upper angles of the uterus; each passes in a curved direction to the internal inguinal opening, through the inguinal canal, and ends in the cellular tissue outside the external abdominal

ring; and each is in its course enveloped in a fold (the anterior) of the broad ligament. They prevent downward and backward displacement of the uterus, a circumstance which explains the rationale of the Alexander-Adams operation of shortening these ligaments in cases of inveterate prolapsus uteri. The broad ligaments (ligamenta lata) are two double layers of peritoneum which pass outwards from the uterus to the lateral pelvic wall; perhaps it would be more correct to describe them as the mesentery of the uterus, for that organ really lies between the folds of peritoneum of which the ligaments are composed; at any rate a part of each ligamentum latum forms a mesentery for the Fallopian tube (mesosalpinx). The base of the ligament may be represented as a wavy line passing outwards to the lateral pelvic wall at a point just in front of the sacro-iliac synchrondrosis; the upper (free) margin passes from the upper angle of the uterus outwards to a point in the ilio-pectineal line midway between the sacro-iliac joint and the pectineal eminence; the inner attachment is along the lateral border of the uterus from the upper angle almost to the lateral part of the roof of the vagina; and the outer edge is attached to the pelvic wall as low down as the level of the ischial spine. Three folds of the broad ligament have been recognised: an anterior, in which lies the round ligament (ligamentum teres); a middle, the mesosalpinx, in which are situated the Fallopian tube and the parovarium and vessels and nerves; and a posterior, to which is attached the anterior margin of the ovary. The utero-sacral ligaments are two muscular bands which pass backwards from the upper part of the cervix uteri to the sacrum, and enclose the rectum on the way; in the anterior part of their extent they raise up the peritoneum of the pouch of Douglas into two folds, the recto-uterine ligaments, which serve to subdivide the pouch into compartments. The utero-vesical ligaments are simply peritoneal folds which pass from the cervix to the posterior surface of the bladder and mark off a slight depression, the utero-vesical pouch. The ovarian ligament and the infundibulo-pelvic have already been described.

Intermediate Organ: the Vagina.—By means of the musculo-membranous canal, the vagina, the internal genital organs are put in communication with the exterior; it may therefore be termed the intermediate organ. It forms a slit in the pelvic floor, lined with mucous membrane, extending from the hymen to the cervix uteri, and lying between the urethra and bladder in front, and the rectum and perineum behind. With the woman in the erect posture the vagina makes an angle of about 60° with the horizon, and is therefore nearly parallel to the plane of the pelvic brim. When the bladder is empty the normal axis of the vagina forms with the long axis of the uterus a right angle; but when the bladder is distended the angle becomes more or less obtuse. When the rectum is full the vaginal axis becomes almost perpendicular. Normally the vaginal walls are in contact, and a sagittal section of the pelvis shows it as a linear slit, while a transverse section reveals an H-shape; it is correct to describe the vagina as a tube only when the finger or instruments are being passed up it, or when the fœtus is being propelled down it. A cast of the distended vagina has the form of a truncated cone, base uppermost. It has two walls, a lower or vulvar end, and an upper end, roof, or fornix. The anterior wall is the shorter, measuring 5 cm.; the posterior has a length of 7·5 cm. The anterior wall has a somewhat triangular shape with the base above and the apex at the vaginal orifice; and its surface is thrown into numerous folds, there being one median longitudinal ridge, the anterior column, and many transverse and oblique rugæ running off from it; these rugæ are continued on the inner aspect of the hymen; and they disappear to a large extent after parturition. On the posterior wall the rugæ show a similar but less marked arrangement; this wall also has a triangular shape, but instead of being straight, as is the anterior, it has a sigmoid curve; like the anterior wall it is reflected upon the cervix forming part of the vaginal roof or fornix. The lower end of the vagina opens at the vulva between the labia, situated laterally, the hymen behind, and the vestibule in front; it is an antero-posterior slit. The roof of the vagina through the projection of the cervix uteri into it is divided in imagination into four parts, called the anterior, posterior, and lateral fornices or fossæ. These fornices, of course, only exist when the vagina is distended; the posterior is much the deepest. The relations of the vagina to surrounding parts are of considerable importance. Anteriorly it is connected in its upper half with the basfond of the bladder, and with its neck by dense areolar tissue which forms the vesico-vaginal septum; in its lower half it is intimately united to the urethra, which is indeed embedded in it, the two together forming the urethro-vaginal septum. Posteriorly the vagina in its upper one-fourth is separated from the rectum by the pouch of Douglas; in its middle two-fourths it is connected by loose connective tissue with the rectum; while in its lower fourth the perineal body lies between it and the rectum and anus. It is important to remember the relation of the posterior fornix to the peritoneal cavity, and it is noteworthy that sometimes the pouch of Douglas is deeper than normal, and may descend between the vagina and rectum almost to the perineum. Laterally the vagina is related to the levatores ani muscles, and to the large venous plexuses. The lateral fornices are related to the bases of

the broad ligaments and to the vessels lying in them. The anterior fornix is distant a little over an inch from the bottom of the vesico-uterine pouch of peritoneum; to its outer sides and well above it are the ureters (Fig. 8).

The structure of the vagina as seen by the naked eye consists of an outer layer or coat of connective tissue and elastic fibres, by means of which it is united to surrounding structures, and in which lie large venous plexuses; of a middle or muscular coat in which separate layers of fibres have been described, concerning the arrangement of which, however, there is a want of unanimity among anatomists; and of an inner layer or mucous membrane, thrown into folds and situated upon a submucosa, of a pink colour usually, but becoming purple in pregnancy. Microscopically the muscular coat

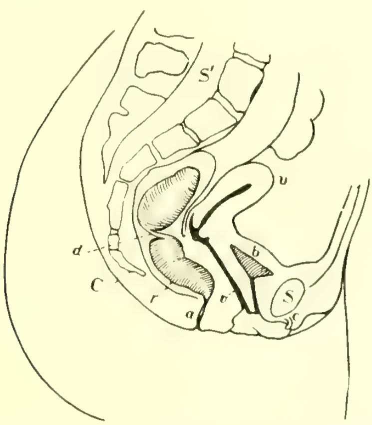


FIG. 8.—Diagram of a supposed mesial section of the pelvis of a living woman: *a*, anal canal; *r*, rectum; *v*, vagina; *c*, clitoris; *b*, bladder when collapsed; *u*, uterus; *d*, valve of rectum (Houston); *S*, symphysis pubis; *S'*, sacrum; *C*, coccyx. (After Dorland.)

is found to consist of unstriped fibres running in bundles in various directions, and between which lie large veins and lymphatics. The submucosa consists of loose areolar tissue which enters into the formation of the columns, and on this lie several layers of pavement epithelium; there are many papillæ and only a few true secreting glands. The mucous membrane, if exposed continuously to the influence of the air, as in prolapsus, becomes skin-like, and it is in many respects a cutaneous rather than a mucous structure. If it be true that it is developmentally derived from the lower ends of the Wolffian ducts (Wolffian bulbs of Hart) which are epiblastic structures, this peculiarity is largely accounted for. The vagina itself is developed from the Müllerian ducts (fused), but it is quite possible that its lining membrane is, as Hart maintains, a derivative of the Wolffian bulbs. In the fœtus the vagina is often distended with much desquamated epithelium, and in vertical mesial section looks as if it were almost the only pelvic content. Great discussion has recently taken place as to the micro-organisms of the vagina. The consensus of opinion now seems to be that normally no pyogenic cocci are present, although many bacteria are to be found, including the thick bacillus vaginalis of Döderlein. The arterial blood-supply of the vagina is from the vaginal arteries (from the internal iliacs), from branches of the uterine arteries, and from branches from the pudendal arteries. An azygos branch in the anterior wall joining the circular artery of the cervix has been described by Hyrtl. The veins, without valves, form complete sheaths for the canal, both outside the muscular coat and beneath the mucous membrane. The lymphatics from the lower fourth of the vagina join those from the external genitals, and pass to the inguinal glands; those from the rest of the canal go to the internal iliac glands. The nerves are from the inferior hypogastric plexus of the sympathetic, from the fourth sacral and from the pudic nerves.

External Generative Organs.—The external genitals collectively constitute the vulva or pudenda, and consist of the labia majora and minora situated laterally, and of the mons veneris, clitoris, vestibule, meatus urinarius, vaginal orifice, hymen, fossa navicularis, fourchette, and perineum in the middle line. The *mons veneris* is an irregularly triangular area of skin with underlying adipose and connective tissue situated in front of the symphysis pubis. It is covered from the time of puberty onwards with crisp, curly hair which rarely passes upwards towards the umbilicus, and in this respect therefore differs from the hair of the same region in the male. Numerous sebaceous and sudoriparous glands are found in the skin of the mons, and in its substance also the external ends of the round ligaments terminate in the form of scattered muscular fibres. From the sides of the mons two folds of skin pass backwards towards the perineum, forming in their course the lateral boundaries of the vulva; these are known as the *labia majora*, or *externa*, or alæ majores. Each labium has on cross-section a somewhat triangular form, the base rests on the pubic ramus, and the sides are the external and internal surfaces. The external surface is covered with rugæ, and has an appearance recalling that of the scrotum in the male; it carries numerous hairs from the age of puberty onwards, and there are plentiful sebaceous glands. On the inner surface the hairs are scanty, and the colour is less dark than that of the outer aspect. In multiparous women and in the old or emaciated the labia are pendulous, and the vulvar aperture gapes, but in nulliparæ and in young or stout persons they are in contact and conceal the underlying structures. The labia are covered externally with a layer of skin, with numerous hair follicles and sebaceous glands; below this are

fibres of unstriped muscle resembling those of the dartos in the male, and beneath them is a cellulo-adipose layer lying upon an elastic fibrous sac (Broca's sac). Sweat glands are present on the outer aspect of the labia. The labial lymphatics pass to the superficial inguinal glands; the arteries are branches of the external and internal pudic and of the epigastric; the veins go to join those of the bulbs and the vaginal veins; and the nerves are from the internal pudic and small sciatic. Posteriorly the labia fuse insensibly with the skin of the perineum.

The *labia minora* or *interna*, or the *nymphæ*, are two slender folds of skin resembling mucous membrane which lie inside the labia majora, and form the lateral boundaries of the vestibule and of the vaginal aperture. They are directed backwards and outwards, diverging from the middle line. Each labium has a smooth outer surface apposed to the inner surface of the labium majus, but separated from it by a deep groove; an internal surface usually in apposition with the internal aspect of the labium of the opposite side, and showing at its base a white line; a free margin, irregular and fringed; an attached margin arising from the side of the vestibule anteriorly, and from the inner aspect of the labium majus of the same side posteriorly; a posterior end which, according to some anatomists, blends with the labium majus about its middle, and according to others is joined with the corresponding part on the opposite side to form a thin fold lying in front of the perineum and called the *fourchette*; and an anterior end which bifurcates at the level of the clitoris, and one part goes above the clitoris to unite with that of the opposite side to form the prepuce, while the other passes below the clitoris to meet that of the opposite side in the frenulum. The nymphæ contain no adipose tissue and do not carry hairs, otherwise they have the structure of skin. The sebaceous glands open alone on the surface. The nymphæ have the same vascular and nervous supply as the labia majora. The *clitoris* is a small curved body with a prepuce and frenulum formed from the anterior ends of the labia minora in the way that has been described above. It lies in the middle line at the apex of the vestibule, and is the homologue of part of the male penis. It consists of the glans, a mass of erectile tissue not larger than a pea and covered with fine skin, and of the body or corpus clitoridis, a cord-like structure which passes to the anterior edge of the pubic arch, where it divides into two crura, the inner margin of each of which is covered by the erector clitoridis muscle. The nerves of the clitoris are very numerous, and end in end-bulbs, and also in the peculiar genital corpuscles of Krause; they come from the pudic. The arterial supply consists of the terminal branches of the internal pudic, a dorsal twig to the glans and prepuce and profunda branches to the corpora cavernosa. There is a dorsal vein which ends in the vesical plexus; the lymphatics pass to the inguinal glands.

The *vestibule* is a triangular area with at its apex the clitoris (just described), with its sides formed by the inner margins of the labia minora, and having for its base the upper margin of the vaginal orifice. At the middle of the base is the meatus urinarius, and surrounding the meatus and stretching up from it towards the clitoris is a band known as the male vestibular band. This band is not always seen in the adult, but is fairly evident in the new-born infant. Under the mucous membrane of the vestibule some veins are found running transversely from one side to the other: these form the pars intermedia, and join together the two vestibular or vaginal bulbs which are placed at the sides of the vestibule. The surface is covered by several layers of pavement epithelium, below which are papillæ with capillary loops, and beneath them a connective tissue layer; there are compound racemose glands. The *meatus urinarius* is the external orifice of the urethra, and is a small dimple in the mucous membrane of the vestibule. The urethral canal at this point is a small vertical slit; immediately within it are the openings of two short tubes (Skene's tubules) which lie in the muscular wall of the urethra, and which are said by some to be relics of Gartner's ducts derived from the lower ends of the Wolffian ducts, and by others to represent the male prostate. Round the meatus are little openings, the orifices of the mucous glands of the vestibule (glandulæ vestibulares minores) already referred to. The meatus lies from 2 to 2·5 cm. below the clitoris.

The *orifice of the vagina* lies immediately behind the vestibule and in front of the fourchette, which, it will be remembered, was formed by the posterior union of the labia minora. It is an antero-posterior slit, and in the virginal condition is partly occluded by the hymeneal membrane. Between the hymen and the fourchette is a small depression to which the fanciful name of fossa navicularis has been given. The *hymen* has usually a crescentic or semilunar shape, but occasionally it is a diaphragm with a single central aperture, or with two apertures, or rarely with several openings in it. It presents a free border towards the vaginal orifice, which is thin and concave, an attached border posteriorly, an inferior or external surface, and a superior surface on which the vaginal rugæ can sometimes be traced. It is composed of connective tissue with arteries and veins in it and many elastic fibres. It is usually but not invariably torn at the time of the first coitus, and it is certainly broken up when the first child passes through the vaginal orifice. Portions of the hymen remain as little fleshy projections (three to six in number), which are

known as the carunculæ myrtiformes. Behind the hymen are the *fossa navicularis* and the *fourchette*, which have been already described; it may be noted that the fourchette is very often torn in the first parturition. Behind the fourchette is the skin covering the base of the perineal body. Reference in passing has been made to the *vaginal* or *vestibular bulbs*. These are elongated masses of veins lying at the sides of the vestibule and vaginal orifice in close relation with the constrictores vaginæ muscles. They are about the size of a bean, but in an injected condition have a length of about 4 cm.; they have a somewhat conical form, and their apices lie at the level of the meatus urinarius, where they communicate by the pars intermedia, while their bases are situated opposite the lower third of the vaginal orifice; and they consist of complicated venous plexuses in a fibrous sheath. They communicate freely with surrounding veins. In addition to the sudoriparous, sebaceous, and mucous glands of the vulva already referred to there remain for description two structures known as the *vulvo-vaginal glands*, glandulæ vestibulares majores, or the glands of Bartholin, Duverney, or Méry. These are two small oval bodies, having the size of a bean or an almond, and situated on either side of the vaginal orifice near the posterior ends of the vaginal bulbs. They lie beneath the superficial perineal fascia, are racemose, mucous, or sero-mucous glands, consist of numerous acini lined with columnar epithelium, and discharge their secretion by an excretory canal which opens at the point of union of the posterior third with the anterior two-thirds of the vaginal orifice in the furrow separating the hymen from the internal surface of the labium minus.

The clitoris is the representative of the genital tubercle of the embryo, and the labia are the edges of the genital cleft. Behind the genital tubercle there is in the early stages of development a cloaca common to the urogenital canal and to the rectum; but through the growth of a septum which becomes the perineal body the rectal canal is separated from the space in front known as the urogenital sinus. Later the vesico-vaginal septum divides the urogenital sinus into two parts, the urethra and the vagina. According to some, the hymen is simply the lower end of the vaginal mucous membrane everted; according to others, it is formed from the external genitals; and according to Hart, it is due to the breaking down of the Wolffian bulbs from which the vagina receives its epiblastic lining.

The muscular and connective tissues lining and bridging across the pelvic cavity now fall to be described. The pelvic peritoneum with its pouches and duplicatures has been considered, but it is necessary to refer to the muscular structures which lie below it and modify by their presence the diameters of the osseous pelvis. The iliacus muscle on each side lessens the depth of the internal iliac fossa; while the psoas muscle and the vessels near it change the shape of the pelvic brim and diminish the transverse diameter of the brim by about 1 cm. The iliacus and psoas muscles together increase the depth of the pelvic cavity. It may also be noted here that the presence of the rectum causes the left oblique diameter at the brim to be a little less than the right. The obturator internus and pyriformis muscles narrow the transverse and the oblique diameters of the cavity, while the bladder, urethra, and rectum diminish its antero-posterior diameter. The most important soft parts of the pelvis, however, are those which fill up the pelvic outlet and form the pelvic floor or diaphragm; to the description of these some space must now be given.

The pelvic floor is composed of skin, mucous membrane, fasciæ, muscles, and fat; into its composition it may be counted also that the bladder and rectum enter; and although it forms a compact diaphragm it is nevertheless a diaphragm traversed by clefts or lines of displacement, represented by the vaginal and urethral canals. The floor may be studied first by dissection, and second by sections; in this way its dissectional and its structural anatomy are both brought out. On the inner aspect of the pelvic floor lie the uterus, the Fallopian tubes, broad ligaments, ovaries, and the pelvic peritoneum; and on its outer aspect lie the external organs of generation; all these structures have been described. The dissection of the floor is usually undertaken from without inwards. Its skin surface shows a median depression consisting of an anterior cleft in which lie the external organs of generation and a posterior or natal cleft in which is situated the anal aperture. The anus is about 1 inch in length, and is parallel to the axis of the brim of the pelvis, and is in consequence at right angles to the axes of the vagina, rectum, and urethra, for these are all parallel to the plane of the brim. It lies about 4 cm. behind the hymen or fourchette, being separated therefrom by the skin over the perineal body. It has an internal and an external sphincter. The part of the pelvic floor which projects beyond the plane of the outlet of the osseous pelvis is termed the *pelvic floor projection*; it measures about 3 cm. Below the skin of the posterior part of the pelvic floor are situated the superficial fascia, and on each side the base of the ischio-rectal fossa. This fossa has an outer wall formed by the obturator internus, an inner and upper wall composed of the levator ani and the anal sphincter; its apex is the point of junction of these two muscles, and its base lies between the transversus perinei and the edge of the gluteus maximus. It contains much fat. Under the skin of the anterior part of the floor lies the

superficial fascia with its deep layer attached to the pubic arch, and passing round the transversus perinei to join the anterior layer of the triangular ligament. When the fascia is dissected off the perineal muscles three pairs are revealed. They form on each side an isosceles triangle with base posterior; the base is represented by the transversus perinei arising from the ramus of the ischium and inserted into the perineal body; the outer side is formed by the erector clitoridis which passes from the ischial tuberosity to the crus clitoridis; and the inner side is constituted by the bulbo-cavernosus or constrictor vaginæ which arises in the centrum tendineum of the perineal body, and along with the corresponding muscle of the opposite side forms a sort of sphincter for the vaginal orifice. Below the posterior end of the bulbo-cavernosus lies the gland of Bartholin, and farther forwards and also below the muscle is the vaginal bulb. Another muscle which finds an attachment in the perineal body is the external sphincter of the anus. In this place a few words of description regarding the perineal body are necessary. It is found on dissection to be a somewhat pyramidal structure filling up the space between the anus and the vagina, which is produced by the fact that these two canals diverge from one another, the vaginal axis passing forwards and the anal backwards. It is composed of muscular origins and insertions and of fibrous and elastic tissue. The muscles passing into it are the sphincter ani, the two transversus perinei muscles, the two bulbo-cavernosi, and the levatores ani. Its base is covered by the skin between the anus and the vaginal orifice, and constitutes the part torn in perineal lacerations, its anterior side is related to the posterior wall of the vagina, its posterior side to the anterior rectal wall and anus, while laterally there is adipose tissue. Its vertical measurement is about 3·5 cm., and its antero-posterior about 2·5 cm. The structural integrity of this body is an important factor in the solidity of the pelvic floor, for when it is seriously damaged prolapse of the vaginal walls and uterus takes place. If the muscles which have been described be now removed the anterior layer of the triangular ligament is exposed; behind it are the pudic vessels and nerves and the posterior layer of the triangular ligament. Neither the perineal muscles, however, nor the triangular ligament constitute the most powerful supporting elements of the pelvic floor; these are supplied by the levatores ani and the coccygei muscles. The levatores ani have the form of a horse-shoe open in front, and sling the vagina and rectum across the pelvic cavity; they arise from the back of the pubic bone, from the pelvic fascia along the white line, and from the ischial spine, and they are inserted into the rectum, vagina, tip of the coccyx, and into each other. Each coccygeus muscle arises from the spine of the ischium and the inner surface of the pelvic fascia, and is inserted into the coccyx and the lower part of the sacrum; it supplements the levator ani. The bladder, rectum, retro-pubic fat, along with blood-vessels and nerves and the pelvic fascia, make up the rest of the pelvic floor. The pelvic fascia is a somewhat complicated structure. It is continuous above with the iliac fascia, and at a point a little below the level of the brim, called the white line, it divides into a layer which covers the obturator internus (obturator fascia), into a thin sheet which lies on the under surface of the levator ani muscle (anal or ischio-rectal fascia), and into the great visceral or recto-vesical layer which covers the upper surface of the levator ani and passes to the bladder, vagina, and rectum, where it divides into four layers—vesical, vesico-vaginal, recto-vaginal, and rectal. The layers of the triangular ligament to which reference has already been made are connected with the obturator and recto-vesical divisions of the pelvic fascia.

The remaining component parts of the pelvic floor and the relation of the parts of the floor to each other are well brought out by the sectional method of study. A vertical mesial section of the pelvis and its contents shows that the pelvic floor is made up of two segments divided by the slit of the vagina which runs parallel to the plane of the brim (Fig. 8). Hart has given to these segments the names of pubic and sacral. The pubic or anterior segment is composed of the urethro-vaginal septum and the urethra, of the bladder, and of the pad of fat lying between the bladder and the posterior surface of the symphysis pubis, and called the retro-pubic fat; it is somewhat triangular in form; it is loosely attached to the pubic bones; and it is bounded posteriorly by the vagina and in front by the symphysis. It may be called the displaceable segment of the floor. The posterior or sacral segment is composed of the posterior vaginal wall, of the perineal body, of the muscles and connective and fascial structures surrounding the rectum and anus, and of the rectum itself; it is roughly quadrangular in form; is firmly attached to and dovetailed into the sacrum and coccyx, and it is bounded in front by the vaginal slit, and behind by the posterior pelvic wall. It may be called the fixed segment of the pelvic floor. In the resting condition of the floor the two segments are in close contact along the line of the vaginal cleft, for intra-abdominal pressure presses the pubic against the fixed sacral segment, and so produces a supporting floor on which rests the uterus and annexa and the intestines. It has sometimes been stated that the uterus should be regarded as a part of the floor, but it is undoubtedly more correct to regard it as a viscus resting on the anterior segment of the floor. Under certain conditions the segments can be

separated. Thus in prolapsus of the uterus the floor is weakened by general laxity of tissue, or more often by actual destruction of the supporting sacral segment, with the result that the anterior segment with the superposed uterus passes downward past the sacral segment and protrudes from the vulva. In labour, on the other hand, the anterior segment is pulled up into the abdomen, the posterior is pushed backwards, and in the space between the fœtus descends on its way out of the abdomen. Again, if the patient be placed in the genu-pectoral posture, and the vaginal orifice kept open (as by a Sims speculum), the vaginal cleft becomes a cavity, and the cervix uteri is seen to be at a greater distance from the vulva; in this case also the segments of the floor have separated, the anterior passing towards the diaphragm; and this peculiarity has been utilised in the examination of the pelvic viscera and in the treatment of morbid conditions of the uterus and vagina. Yet again, when the volsella is attached to the cervix uteri and downward traction made in the axis of the vaginal cleft, the uterus can be drawn to the vulva without the use of force and examined or operated on; here the anterior segment of the floor has been pulled down past the sacral.

The Bladder and Rectum.—In order to complete this survey of the female genital organs and of the structures in immediate connection with them, it is necessary that the *bladder* and *rectum* be described. The bladder when empty usually forms with the urethra a **Y**, but occasionally its cavity forms a slit continuous with the urethra, and the mucous membrane is thrown into folds; the former is the diastolic and the latter the systolic empty bladder. Further, in the adult woman it is a pelvic content when empty, but in the infant and fœtus it normally lies above the plane of the brim. The upper part alone is covered with peritoneum in the adult. Its wall is composed of several layers of unstriped muscle, and internal to these is a mucous membrane consisting of connective tissue lined by several layers of transitional or multiform epithelium. The mucous and muscular coats are separated by a loose submucosa. There are three openings into the bladder, the internal orifice of the urethra and the orifices of the two ureters. It is necessary, in connection with the surgery of the genital organs, to remember the course of the ureters in the pelvic cavity; each ureter having crossed the iliac vessels passes downwards, backwards, and a little outwards on the wall of the pelvis to a point near the ischial spine, where it bends downwards, forwards, and inwards, and passes beneath the base of the broad ligament, and is crossed by the uterine artery about the level of the os uteri internum; it then passes at the side of the upper third of the vagina, runs for a distance of about 1·5 cm. in the vesico-vaginal septum, and then traverses the bladder wall in a direction obliquely downwards and inwards to open at one angle of the trigone. The urethra is a straight slit measuring about 4 cm. in length. It is parallel to the vagina, and is indeed embedded in its anterior wall. It is lined with a mucous membrane covered with squamous epithelium in its lower part, and with transitional epithelium in its upper part; it has also a muscular coat and a submucosa. Its external orifice with Skene's tubules has been already described.

The rectum extends from the left sacro-iliac joint to the anus, and is divided into three portions. The first has a mesentery (mesorectum) and ends at the third sacral vertebra; the second part shows reflection of the peritoneum on to the upper part of the vaginal wall; and the third has no relation to peritoneum at all, and lies behind the posterior vaginal wall, separated from it by loose tissue and lower down by the perineal body. It is lined with a mucous membrane covered with columnar epithelium and showing numerous Lieberkuhnian follicles. There are oblique folds consisting of the mucous membrane of the submucosa, and of some circular muscular fibres; one of these lies near the level of the sacrum, another about 4 cm. above the anus, and a third intermediate. The lowest of the three is called the sphincter tertius or valve of Houston (Fig. 8). In the lower part of its extent the rectum runs parallel to the vagina, but the anal canal turns sharply backwards and is at right angles to the vaginal axis.

Arrested Developments of

See also Hermaphroditism.

The *malformations of the female genital organs* are mostly instances of arrested developments, that is to say, in them we see conditions which are temporary in normal organogenesis and which have become permanent. For instance, the uterus which is originally composed of two lateral halves (the Müllerian ducts) is normally an organ which shows no trace of its temporary duplicity, but which may, under certain circumstances, exhibit signs of it, as is evident in the various types of "double uterus." In some few cases this comparatively simple explanation of genital malformations fails, as in the rare anomaly known as trifid uterus and in double vulva; but, speaking generally, a malformation of one or other of the genital organs represents a state of affairs which at an early stage of development would be normal.

Malformations of the ovaries have an important bearing upon the fertility and health of the individual who is the subject thereof, for the ovary is not only the gland which forms the

ova, but it is also in all probability a structure which secretes a product without which perfect health is impossible. *Complete absence* of both ovaries is rare except in monstrosities, even *unilateral absence* is uncommon; but a rudimentary state of the glands is more frequent. *Rudimentary ovaries* show only a small number of ill-developed ovisacs in a considerable quantity of connective tissue; and they are not uncommonly displaced into the inguinal canals, or lie at a higher level than is normal. The clinical importance of these defective conditions depends greatly upon their unilateral or bilateral character; in the former case reproductive activity may be little interfered with; in the latter the secondary sexual characters will be absent, the menstrual function will not be established, and sterility will be inevitable. There may also be associated morbid states, such as epilepsy, chlorosis, and the like. The diagnosis is often impossible without a laparotomy, although there may be a strong presumption in favour of defective ovaries. It has to be borne in mind that the uterus may be normal. *Accessory* or, more correctly, *constricted ovaries* are to be found in from two to three per cent of autopsies: they are about as large as a pea, and are generally attached to the normal ovary by a pedicle; they may contain Graafian follicles, and are due either to fœtal peritonitis causing separation of a piece of the ovary or to budding of the primitive sexual gland; and their presence probably accounts for the persistence of menstruation or the occurrence of pregnancy after a double ovariotomy or oöphorectomy. *Congenital hernia of the ovary* is nearly always inguinal, for, through the persistence of the canal of Nuck, the gland passes down into the inguinal canal and may reach the labium majus; in such cases the swelling in the groin is subject to periodic monthly enlargement, a circumstance which makes the diagnosis of its nature possible. It may give rise to much pain and to reflex phenomena, and must then be excised. It is often associated with the uterus unicornis and hernia of the Fallopian tube, as has recently been brought out by Browne (*Trans. Amer. Gyn. Soc.* xxiii. 352, 1898).

The *malformations of the Fallopian tubes* can hardly be diagnosed during life save by the opening of the abdomen. Both tubes may be *absent*, but more commonly *one alone is wanting*, and then it is frequently found that there is also a uterus unicornis and unilateral absence of the ovary. The tubes also may be present in a *rudimentary* or *solid condition*, an anomaly which is practically equivalent to their absence. The *spiral convolutions* which normally exist in antenatal life may persist as abnormal twists in adult life; possibly they may be the causes of dysmenorrhœa and sterility. *Accessory tubal ostia* and tufts of fimbriæ are not uncommon (three to six per cent); as many as six have been seen in one case; they are usually situated near the outer end of the tube; and it has been thought that they may sometimes be the cause of extra-uterine gestation.

The *malformations of the uterus* may be divided into those showing apparent excess in formation, those due to defect, and those due to displacement. With the exception of the extremely rare *trifid* or *accessory uterus*, the various forms of *double uterus* are only apparently due to excessive formation; they are really defective developments, being caused by more or less complete want of fusion of the portions of the two Müllerian ducts which normally unite to make the single uterus. The most complete want of fusion is seen in the *uterus didelphys, s. duplex.* In it there appear to be two single uteri lying side by side, but each possessing only one ovary and tube; there may also be two vaginal canals and a double opening in the hymen. The *uterus bicornis* is a much commoner type; in it the upper part of the uterine body is evidently separated into two horns or halves, while the lower part of the body and the cervix are single; in its most marked form the two horns are wide apart, and between them passes a band (recto-vesical) from the bladder to the rectum; but in the slightest degree there is only a notch or groove at the fundus to indicate externally the double nature of the organ (*uterus cordiformis*). One or both horns may be imperforate, and the cervical canal may be double or single. In its external appearances the *uterus septus* or *bilocularis* gives no indication of its double character, yet it is divided more or less completely into two cavities by an internal septum running usually antero-posteriorly. The septum may be present in the whole extent of the organ from fundus to external os, or it may be absent above and present below, or *vice versa*, or yet again the septum may be incomplete in the middle. There may also be a certain degree of rotation of the uterus so that the cavities come to lie antero-posteriorly. All the forms of double uterus are difficult of diagnosis, the conditions with which they are most apt to be confounded being fibroid tumours and extra-uterine pregnancy. The occurrence of a regular menstrual discharge every fortnight will excite suspicion, and then the physical examination may reveal a double cervical orifice, or the sound may pass into a double cavity; but many times the malformation has not been discovered till labour has come on and called for manual interference, for the double uterus does not entail sterility, although it may predispose to malpresentations. Pregnancy may occur in one half of the double uterus and menstruation may continue from the other, or decidual membranes may form in it; pregnancy may also occur in both horns simultaneously, or at different but not widely separate dates; or again the two horns may become

impregnated alternately. When the half in which the fœtus lies is rudimentary in its development the case is clinically one of extra-uterine gestation.

The *uterus unicornis* is due to the absence of one Müllerian duct; sometimes, however, the name is also given to the anomaly in which the second horn is not altogether absent, but exists as a solid or hollow rudiment, representing an imperfect development of one Müllerian duct. The uterus unicornis inclines to one side, and tapers to a point which is continuous with the Fallopian tube, and is marked by the attachment of the round ligament. The cervix and vagina are usually small and poorly developed. The diagnosis is rarely made during life, for menstruation and even pregnancy may occur normally; but pregnancy in the rudimentary horn attached to the uterus unicornis is a serious circumstance, having all the prognostic significance of ectopic gestation, rupture usually taking place into the peritoneal cavity.

The term *uterus rudimentarius* has no very precise pathological signification, for it has been applied to the cases in which the organ was reduced to a strip of muscular tissue lying between the layers of the broad ligament (*uterus membraniform* or *membranaceus*), to those in which there was a fair-sized fibro-muscular uterus, but without a cavity or with a very incomplete one (*uterus solidus*, *uterus partim excavatus*), and to those in which the fœtal or the infantile characters of the organ were retained in adult life (*uterus fœtalis*, *uterus pubescens*. The *complete absence* of the uterus in an individual otherwise female is a very rare occurrence, and the condition diagnosed clinically as absence of the uterus (*uterus deficiens*) is generally one or other of the forms of rudimentary uterus. In all these conditions there are often found to be other defects of the genital apparatus, such as imperforate vagina, rudimentary ovaries, ill-developed mammary glands, and absence of hair on the mons. Clinically there is generally amenorrhœa, but in the fœtal and infantile types there may be a scanty and painful discharge; there is always sterility, and commonly dyspareunia; and should normal ovaries be present there will be so much periodic pain as to necessitate the consideration of the question of their removal. The diagnosis of the anomaly can only be made by a careful vesico-vagino-abdominal examination of the pelvic contents; in the absence of a perforate vagina a recto-vesico-abdominal examination will be required. In the case of the fœtal and infantile uterus attempts to stimulate growth by means of ferruginous tonics, intra-uterine stem pessaries, and electricity, have been made and occasionally have obtained a certain measure of success: but in the more marked forms of the rudimentary uterus the removal of the ovaries is generally indicated if the sufferings of the patient from ovulation are severe.

Congenital anteflexion and *retroflexion* of the uterus have been regarded by some as malformations; it is probably more correct to look upon them as the results of fœtal disease (fœtal peritonitis), or of pelvic peritonitis in childhood. They are attended by menstrual suffering (scanty and painful), by sterility or early abortion, and by various reflex phenomena; and are very rebellious to treatment. *Congenital prolapsus uteri* is a very rarely observed condition; so far all the cases recorded (about a dozen) have been accompanied by lumbo-sacral spina bifida which has been the cause of early death.

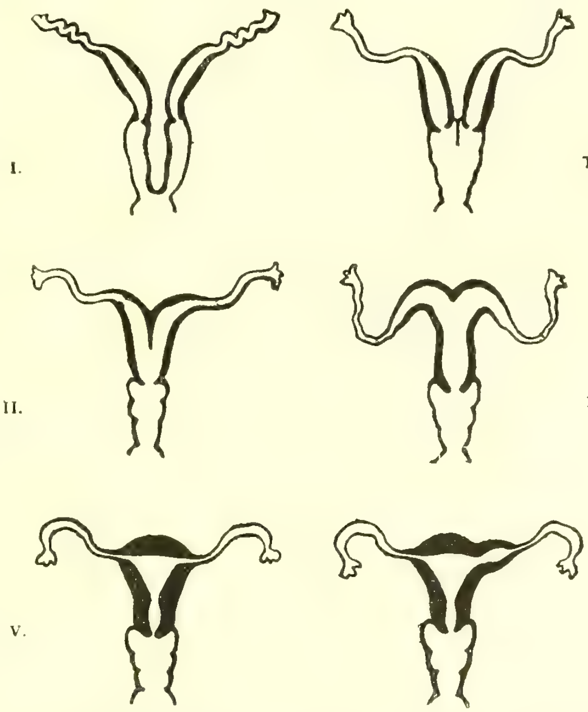


Fig. 9.—Diagram of the tubes, uterus and vagina in different mammals, representing also the malformations which occur in man. I. Double uterus and vagina (some marsupials); II. double uterus, vagina partly divided by septum (rabbit); III. uterus single below but double above (most rodents and cheiroptera); IV. uterus single but with two horns (uterus bicornis of ungulata and carnivora); V. single uterus (man and apes); VI. uterus with rudimentary cornu (uterus unicornis cum cornu rudimentario of man). The uterus unicornis of man is not figured. (Fothergill.)

The malformations of the vagina in many respects resemble those of the uterus. *Double vagina*, for instance, is due, like double uterus, to want of fusion of the Müllerian ducts. A septum, more or less complete, divides the vagina into two lateral canals (rarely into two canals lying antero-posteriorly). In the least-marked degree there is only a ridge on the

vaginal wall or on the cervix to indicate the double nature of the canal. The septum varies much in thickness; it may be absent above and present below (*vagina septa infra*), or present above and absent below (*vagina septa supra*); and it may be perforated in several places. Generally the anomaly is associated with one or other of the forms of double uterus; there may also be a double hymen, but a double vulva is most uncommon, and probably belongs to quite another group of anomalies (double terata). In cases of labour with vagina septa the septum may cause delay and require to be divided, but it may also be pushed aside, and may indeed pass undetected. One of the canals may be imperforate, and mucus may collect in it, giving rise to the erroneous diagnosis of vaginal cyst: when it is associated with a functionating uterus, blood will accumulate in it (*unilateral hæmatocolpos*) and require evacuation.

Unilateral vagina is practically always a concomitant of uterus unicornis. The canal is narrow, and lies somewhat to one side of the middle line; it in fact represents one of the Müllerian ducts, the other having aborted.

Complete absence of the vagina (*defectus vaginæ*) is very rare, and is nearly always associated with advanced teratological conditions, such as sympodia, but incomplete development of the vagina is comparatively common. To this condition the name *vagina rudimentaria* has been given, but a better term is *atresia vaginæ*. There is a complete or an incomplete imperforate condition of the vagina; simply a muscular cord may lie between the rectum and the bladder, or the canal may be present above and absent below, or *vice versa*, or membranous diaphragms may exist in it at various levels. These anomalies are due to imperfect canalisation of the fused Müllerian ducts; but when the lower or vestibular part of the vagina alone is absent it may be supposed that the fault lies, as Hart suggests, in the Wolffian bulbs which normally form the lining of this portion of the canal. The uterus and ovaries may also be imperfectly developed; at the same time this cannot be counted upon, for not infrequently the internal genitals are functionating, and then the condition known as *hæmatometra* (retention of menstrual blood in the uterus) is produced. Clinically, vaginal atresia does not attract notice till puberty, when it is found that menstruation is not established. There may be, however, every month a feeling of weight in the pelvis along with headache, swelling of the mammæ, and epistaxis; and a physical examination may then reveal the absence of the vaginal canal, or (in the less-marked varieties) the presence of a bulging membrane at the vulva. Probably many of the cases of so-called imperforate hymen are really instances of imperforation of the lower part of the vagina, and sometimes the hymen can be seen on the outer aspect of the bulging membrane. By means of rectal touch, aided by a sound in the bladder, the amount of vaginal imperforation can be made out, and the chances of successful surgical interference gauged. It may become necessary to operate, either to allow the escape of retained menstrual blood or to establish an artificial vagina for coition. In the least-marked degree a crucial incision through the membranous obstacle will suffice to set the discharge free; strict surgical cleanliness must, of course, be observed, and means taken to prevent subsequent contraction of the canal. In the more extensive imperforations it becomes very difficult to reach the uterus, and dissection upwards between the bladder and the rectum may be tedious and ultimately futile; the opening of the abdomen in order to operate from the peritoneal aspect has been scarcely more successful. Recently, however, it has been proposed to make straight for the pouch of Douglas, and not to attempt to avoid it, to open into it as in the operation of posterior colpotomy, and then by palpation inside the pelvis to determine whether the uterus and ovaries are healthy. In the presence of functionating internal genitals it will then be well to attempt to form a vagina by invaginating the vulvar mucous membrane, and uniting it with the cervix uteri, and the opening into the pouch of Douglas can then be closed. When the internal organs are not active the gynecologist will be well advised to leave things alone; the demand for an artificial vagina must come from the patient herself, and all the dangers of the operation ought to be pointed out to her and to her husband. By *lateral atresia vaginæ* is meant an imperforate condition of one-half of a septate vagina; blood may accumulate in it and give rise to *lateral hæmatocolpos*; suppuration also may take place in it, *lateral pyocolpos*; in either case the treatment is incision of the sac, with, sometimes, excision of the sac-wall.

Malformations of the vulva do not all admit of so simple an explanation as the anomalies of the uterus and vagina, for they are due to defective development of the posterior end of the embryo, a part whose normal development is not as yet fully understood. The presence of *two vulvar apertures* (one of the rarest of malformations) probably indicates a minor degree of posterior duplicity of the trunk. *Atresia vulvæ superficialis* is not a malformation in the true sense of the term, for it is doubtless due to adhesion of the labia produced by fœtal or infantile vulvitis. It is easily corrected; sometimes simple traction of the labia serves to separate them; in other cases it is necessary to pass a director in at the anterior opening (in the neighbourhood of the meatus urinarius) and cut down upon it. *Vulvar anus* (*atresia ani vaginalis*) is a name given to a condition in

which the normal anus is absent, and the rectum opens into the vagina or vulva (or rather into the urogenital sinus, for the anomaly is really a persistence of the early cloacal state). The chief symptom is the passage of fæces, with or without sphincteric control, in the neighbourhood of the posterior commissure of the vulva. Operative interference, which is most likely to be successful if undertaken when the patient is about fifteen years old, consists in passing a probe in through the vulvar anal opening, and bringing it out at a spot corresponding to the normal position of the anus, and then dividing the parts lying between and bringing the rectum down and suturing it in position. Recently it has been suggested to split the fibres of the levator ani in order to gain a good sphincter. *Vulva infantilis* is of importance only as indicating that in all probability the internal genitals likewise are in an undeveloped or infantile state. *Atresia hymenalis* is a malformation which is presumably often met with, for many cases are on record, but there is reason for believing that many of these cases have really been instances of imperforation of the lower end of the vagina. At any rate the symptoms, diagnosis, and treatment of the anomaly are the same as those of the minor degree of vaginal atresia. The *clitoris* may be *absent*, less rarely it is *split*; both these malformations occur in association with epispadias in women or with ectopia vesicæ. *Hypertrophy* of the *clitoris* and *labia* may be met with as a congenital anomaly, and may give rise to local irritation and to more distant reflex phenomena; excision of the enlarged parts will then be indicated. *Adhesion of the clitoris* to its prepuce and to the nymphæ has much the same symptoms as phimosis in the male, and may be cured by the separation of the adhesions. Finally, it may be noted that the *hymen* may be unusually *fleshy* or *tough*, or that it has an abnormal shape (*labiate, denticulate, fimbriated*, or *circular*). When it is tough it may cause dyspareunia or prevent coitus altogether, and when it is abnormally vascular it may lead to the occurrence of serious hæmorrhage from rupture in coition. Sometimes there are two apertures in the hymeneal membrane (*hymen septus, biforis*), rarely there are several (*hymen cribriformis*).

Genetous Idiocy.—Congenital mental defect in which the pathology cannot be properly diagnosed till after death (Ireland), such as that due to antenatal inflammation of the brain. *See* MENTAL DEFICIENCY (*Primarily Neurotic Type*).

Geneva Lens Measure. *See* REFRACTION (*Measurement of Cylinders*).

Genial.—Relating to the chin, *e.g.* genio-hyoid, genio-glossus, genio-pharyngeus.

Geniculate Bodies. *See* PHYSIOLOGY, NERVOUS SYSTEM, CEREBRUM (*Visual Centre*).

Genital.—Relating to reproduction, *e.g.* the genital canal and the genital organs.

Genito - Urinary. — Relating to the genital and the urinary organs in association; these organs are closely related developmentally and anatomically, and in pathology disease of the one set is often associated with disease of the other. *See* BLADDER; GENERATION, FEMALE ORGANS OF; KIDNEY; LUNG; TUBERCULOSIS (*Complications, Genito - Urinary Tuberculosis*); PENIS; PROSTATE GLAND; SCROTUM AND TESTICLE; URETHRA; UTERUS; VAGINA; VENEREAL DISEASE.

Genoblast.—A sexual or reproductive cell, ovum or spermatozoon.

Gentianæ Radix.—The dried roots of *Gentianæ lutea*, containing an active bitter principle *Gentio-picrin*. *Preparations*—(1) Extractum Gentianæ. *Dose*—2-8 grs.; (2) Infusum Gentianæ Compositum. *Dose*—½-1℥; (3) Tinctura Gentianæ Composita. *Dose*—½-1ʒ. Gentian is a typical bitter, and is prescribed as a stomachic and general tonic, usually in combination with other drugs. It is widely used because it has a pleasant taste and is not so disagreeably bitter as Calumba and other members of this class.

Genu.—The knee. The term is specially used in the description of certain malformations which may affect the joint, *e.g.* genu recurvatum, g. valgum, and g. varum. *See* DEFORMITIES (*Lower Extremity*); KNEE-JOINT, DISEASES AND SURGICAL AFFECTIONS.

Genu - pectoral Position. — The genu-pectoral or genu-facial position is that in which a patient is placed when he (or she) takes up a kneeling attitude, and rests the weight of the upper part of the body on the chest or face and shoulder; the modified genu-pectoral or Sims' position is that commonly used in *Gynecology*.

Geographical Tongue.—A peculiar form of eczema (?) of the tongue in which there are circinate patches of desquamation, which may run into each other and produce outlines resembling those of the continents and islands of a map.

Geophagia.—The practice of earth-eating. *See* PICA OR DIRT-EATING.

George's Calorigen. *See* CALORIGEN.

Gerber's Process.—The estimation of the amount of fat in milk by collecting it on filter paper (free of fat) and extracting by ether.

Gerhardt's Reaction.—The claret coloration of urine when a solution of perchloride of iron is added to it, indicating the presence of diacetic acid. *See* Diabetes Mellitus (*Symptomatology, Urine*).

Gerlier's Disease.—A form of vertigo, accompanied by paresis of the limbs, ptosis palpebrarum, and depression; it is endemic in Switzerland, France, and Northern Japan; endemic paralytic vertigo.

Germ.—A term somewhat widely used to express a microbe or micro-organism (especially one believed to cause a disease), or the rudiment from which an organism develops. The name may be given, in Human Embryology, to the new organism before the first traces of the embryo have appeared in the embryonic area. It is also applied to the rudiment from which any part of an organism develops, *e.g.* a dental germ.

German Measles. *See* Rubella; *also* Fourth Disease; Measles; etc.

Germany. *See* Balneology (*Germany, Waters*).

Germicide.—A substance or influence capable of destroying germs, especially the germs of disease.

Germinal Pathology.—Germinal life ends with the appearance of the rudiments of the embryo in the blastocyst, and it begins (in one sense) with the fertilising union of the spermatozoon and ovum, or (in another sense) with the specialisation of the reproductive cells in the genital ridge. Morbid processes may develop during germinal life as thus defined, and these changes and processes collectively constitute germinal pathology. It is believed that germinal pathology includes, therefore, anomalies in the formation of the blastocyst and of its contained parts, including the earliest rudiments of the embryo, unusual modes of segmentation of the impregnated ovum, errors in the details of impregnation (*e.g.* polyspermy), imperfect maturation of the ovum, imperfect formation of spermatoblasts and spermatozoa, and morbid heredity. If polyspermy be the cause of double monsters, then the large part of Teratology, which deals with double terata, really belongs to germinal pathology also. *See* Ballantyne's *Antenatal Pathology*, vol. ii. pp. 607-659.

Gero-.—In compound words gero- (from the Greek γῆρας, old age) means relating to the aged or senile, such as *gerocomia*, the care of the aged; *geromorphism*, the appearance of old age; and *gerontophthalmia*, or senile ophthalmia.

Gestation.—Pregnancy. *See* Ectopic Gestation; Pregnancy; etc.

Gheel.—A town in Belgium (Antwerp) where the family treatment of insanity has long been in operation ("Colony of Gheel").

Giant.—An individual markedly larger than the average individual of that species, type, and age; one showing a "monstrous largeness" of all his parts. *See* Giantism.

Giant Cells. *See* Physiology, Blood (*Erythrocytes*).

Giant Colon.—Megacolon; enlargement, sometimes congenital, of the colon with hypertrophy of its walls.

Giantism.—A state of "monstrous largeness" of all or of some of the parts of an individual; general, unilateral, or partial macrosomia; it may be antenatal (fœtal macrosomia or giant fœtus) or postnatal, coming on early in childhood or at an advanced period of adolescence. *See* Acromegaly; Hypertrophy (*Giantism*); Teratology; etc.

Gibbosity.—A swelling or protuberance, especially the hump on the back due to spinal kyphosis.

Gid.—A disease in sheep, due to the presence in the brain of the *Cœnurus cerebralis*, and characterised by vertigo. *See* Hydatid Disease.

Giddiness. *See* Vertigo; *see also* Brain, Tumours of (*Symptoms*); Brain, Cysts, etc. (*Progressive Softening, Clinical Features*); Brain, Cerebellum, Affections of (*Hæmorrhage*); Chlorosis (*Symptoms*); Ear, Examination of (*Symptomatology, Vertigo*); Nephritis (*Chronic Nephritis, Renal Cirrhosis, Symptoms*); Stomach and Duodenum, Diseases of (*General Symptomatology*).

Gigli's Saw.—The wire saw recommended by Gigli of Florence, and used for the section of bone as in such an operation as pubiotomy or (more correctly) hebotomy. *See* Labour, Operations (*Symphysiotomy*).

Gilles de la Tourette's Disease.—A disease resembling chorea, in which there are involuntary muscular movements (especially of the face and arms), barking sounds, inarticulate cries, obscene expressions, and mental symptoms resembling those of a monomania; it usually is met with in children, often in those that have a neurotic family history, and it is allied to hysteria; it is also called "impulsive tic."

Gilsland. *See* Balneology (*Great Britain, Sulphur*); Mineral Waters (*Sulphur, Simple*).

Gimbernat's Ligament. *See* Hernia (*Femoral, Anatomy*).

Gin. *See* ALCOHOL (*Spirits*); LIVER, DISEASES OF (*Portal Cirrhosis or Gin-drinker's Liver*).

Ginger. — The dried root of *Zingiber officinale,* found in the East and West Indies. It contains a variety of resins and other bodies, but the most important ingredient is a volatile oil to which it owes its characteristic aromatic flavour. *Dose*—10-20 grs. *Preparations*—(1) Syrupus Zingiberis. *Dose*—½-1 ʒ; (2) Tinctura Zingiberis. *Dose*—30-60 m. The action of ginger depends on the presence of the volatile oil. It is given as an antispasmodic and carminative to allay flatulence, and it is also used as a flavouring agent. It may be added to purgative medicines to prevent griping.

Gingivitis.—Inflammation of the gums; ulitis. *See* MOUTH, DISEASES OF (*Diseases of Gums*).

Ginglymus.—A hinge-like diarthrodial joint, *e.g.* the elbow or the knee.

Ginseng. — A medicine to which the Chinese attribute wonderful remedial powers; it is the root of *Aralia ginseng* and *Aralia quinquefolia*; and its properties are probably nothing more than those of a demulcent.

Girdle-Sensation. *See* SPINAL CORD, MEDICAL (*General Symptomatology, Girdle-sensations*); TABES DORSALIS (*Symptomatology, Objective Symptoms, Trunk Anæsthesia*).

Glabella.—The hairless space at the root of the nose between the two superciliary ridges; also the hairless area over the lower end of the spine above the vertex coccygeus, which is termed *g. coccygea.*

Glabrificins. *See* ANTIBODIES.

Glaisher's Tables. *See* METEOROLOGY (*Hygrometry*).

Gland. — An organ in the human body having as its function the excretion of matters from the economy, or the elaboration from the blood of matters which serve a useful purpose in the economy. *See* PHYSIOLOGY, TISSUES (*Glands*); PHYSIOLOGY, FOOD AND DIGESTION; LIVER, PHYSIOLOGY OF; KIDNEY, PHYSIOLOGY OF; THYMUS GLAND; THYROID GLAND; etc. etc.

Glanders (Farcy).

SYNONYMS.—Gk. μᾶλις or μῆλις; Lat. *Malleus, Equinia*; Ger. *Rotz* (*Wurm*); Fr. *Morve* (*Farcin*); It. *Morva* (*Farcino*); Span. *Muermo.*

See also NOSE, CHRONIC INFECTIVE DISEASES (*Glanders*); PALATE, DISEASES OF (*Glanders*); PERITONEUM (*Pathology, Bacteriology, Glanders*).

I. DEFINITION.—Glanders is a specific infectious disease caused by the *Bacillus mallei,* usually affecting the horse, mule, or donkey, and at times communicated to man and other animals. Where the disease is localised in the nose or internal organs it is called glanders, the cutaneous form being called farcy. The disease runs an acute, subacute, or chronic course.

II. HISTORY.—Glanders has been known from ancient times as a disease affecting horses. It was known to Aristoteles and Hippocrates the Hippiatrist, as also to the Roman writers Apsyrtus and Vegetius (1370-90), the last describing "Malleus humidus" and "farciminosus." Its contagiousness was recognised in the seventeenth century. Solleysel (1664) considered that it could be conveyed through the air, van Helmont (1682) considered syphilis identical with glanders. Saunier (1734) gives directions for disinfecting stables, whilst Garsault (1741) and Bourgelat (1764) recommended the immediate destruction of glandered horses together with the isolation of those suspected. Abildgaard and Viborg in Denmark at the end of the eighteenth century proved that glanders could be communicated by secretions or pus of diseased animals, less frequently was the virus contained in their blood. Virus which had been dried or exposed to a temperature of 45° C. was innocuous. Similar results were obtained in England by Coleman and Delabère-Blaine. Rayer (1837) proved that glanders was communicable to man. The first bacteriological investigations were made by Zürn and Hallier, Christot and Kiener (1868), subsequently by Bouchard, Capitan, and Charrin (1881-82), but it was not until 1882 that Löffler and Schütz isolated the bacillus, and proved finally that glanders or farcy is a specific infectious disease caused only by the *Bacillus mallei.* The last three French investigators had succeeded in cultivating the bacillus from a man and a horse, and, after several generations in

culture, successfully infected donkeys, cats, and guinea-pigs. Working with fluid culture-media, they concluded that the specific agent was a coccus growing in chains. O. Israel (1883) obtained cultures on blood-serum from horses, and successfully infected rabbits with the bacillus, whilst Kitt (1883-84) and Weichselbaum (1885) confirmed and extended the knowledge gained.

III. Geographical Distribution and Prevalence.—Though glanders is not as prevalent as it used to be, nevertheless it is the most dangerous equine disease. It would appear as if glanders can occur in any climate, but it certainly exhibits a predilection for certain localities. Its gradual increase as we go south (the reason of this is unknown) is evident from the figures given by Krabbe for 1857-1873 per 100,000 horses: Norway 6, Denmark 8·5, England 14, Wurtemberg 77, Prussia 78, Servia 95, Belgium 138, France 1130, Algiers 1548. During the years 1877-1887 there were 4 per 100,000 in Sweden (Lindqvist), 300 per 100,000 in Austria. During 1884-1897 in Germany the frequency was 20 - 30 per 100,000. In European Russia glanders is very prevalent, being estimated at 400 per 100,000. In 1898 there were 1380 horses attacked in England (almost entirely in metropolitan districts), 138 in Belgium; there were 657 outbreaks in France and 17 in Denmark. In Hungary the loss from glanders was 39 per 100,000 in 1897; and in the same year Italy lost 277, and Switzerland 58 horses (dead and killed). Sanders (1896) says glanders has been known but recently in South Africa. Dávalos (1894) says it was introduced into Cuba in 1872 (9 cases between 1888 and 1893). Penna (1898) reports it from the Argentine Republic, and v. Velzen from the Dutch East Indies. It also occurs in the U.S. and Canada.

IV. Animals affected.—The disease occurs spontaneously in the horse, mule, donkey, and at times in menageries (lion, leopard, tiger, bear). A number of other animals, have, however, been found susceptible to experimental infection. The difference in the statements of authors regarding the degrees of susceptibility of certain animals to inoculation would seem to depend in part upon variation in the virulence and the amount of the culture used. Highly susceptible are field-mice, *Arvicola arvalis*, *A. terrestris* in Europe, *A. riparius*, *A. austerus* in America, *Spermophilus guttatus* and *S. Citillus* in Eastern Europe, *Spermophilus Townsendi* and *S. Richardsoni* in America, the cat, guinea-pig, hedgehog (*Erinaceus europeus*). White and house-mice are more resistant, though susceptible to virulent cultures. Rabbits, the goat, sheep, camel, and young dog have been successfully infected. The resistance of adult dogs has been overcome by inoculating cultures whose virulence had been raised by intracerebral inoculation in another dog. Young pigs have been successfully infected by inoculation into the anterior chamber of the eye or into the lungs, but not through the skin. Adult pigs, unless weakened, are absolutely resistant, as are also cattle (including the calf), chicken, pigeon, linnet, and frog (Löffler, 1886; Kitt, Weichselbaum, 1885; Csokor, 1888; Peuchu, 1889; Straus, 1889; Preusse, Grünwald, Salmon, Sanarelli, Kranzfeld, Cadeac and Malet, Tartakowski, Dschunkowski, 1886-99).

V. Bacteriology.—The *Bacillus mallei* is found in the affected tissues and organs and their secretions, as also the discharges from ulcers, etc. They may be very generally distributed in the body. In man, the horse, sheep, guinea-pig, and rabbit, they have been isolated and cultivated from nodules, cutaneous abscesses, the greatly enlarged skin follicles, ulcers of the mucous membranes, more rarely from marrow and blood. They have been found in the saliva and milk, but still oftener in the urine secreted by kidneys showing no appreciable lesion. In man they have been found in the abscesses and blood (few) during life. In the horse they may appear in the blood during a febrile attack or after a mallein injection. They are more numerous in the blood in acute cases. It is often difficult to find the bacilli microscopically in the nodules, but their presence may be demonstrated by cultures or inoculations upon animals (Löffler, Weichselbaum, Ferraresi and Guarnieri, Lissitzin, Preusse, Babes, Hallopeau and Jeanselme, Bonome, Duval, Gasne and Guillemot, Noniewitz, Kitt, Trambusti, Philipowitcz (1886-98).

Morphology.—The form of the glanders bacillus varies greatly. The bacilli measure 1·5-3 by 0·25-0·4 μ. They have rounded extremities, and are often slightly curved. Noniewitz (1891) claims to have observed differences in the form of bacilli within the body. In acute cases well-formed bacilli are encountered, in subacute and chronic cases more highly refractive coccus-like bodies are found, which suggests that many of them degenerate in the body. Gutzeit (1892), as also Johne, finds the bacilli are larger when grown in horse-bouillon than when cultivated in beef-broth. Already Löffler observed club-shaped, filamentous, and coccus-like forms in culture, and this has since been seen and described by Kranzfeld, Levy, Semmer, Marx, and Galli-Valerio (1887-99); the two latter, as also Conradi (1900), have in addition observed true branching in organisms grown in culture. Lubarsch (1899) was unable to observe branched actinomyces-forms in rabbits which had been inoculated into the kidney or subdurally. The glanders bacilli do not form spores. Refractive bodies which have been considered to be spores by Baumgarten (1888) are almost certainly

degenerative. These bodies show no greater resistance to heat and chemical agents than do the ordinary bacilli, and nobody has claimed to see bacilli issue from them. The long thread-like forms may measure 10·26 μ or more. The bacillus is non-motile.

Staining Reactions.—Smear preparations should be made as thin as possible. The bacillus stains readily with basic aniline dyes, presenting usually irregularities, *i.e.* alternate deeply staining or clear and faintly staining parts. Bacilli are best looked for in young nodules. Owing to the fact that they readily decolorise and are often hidden by the nuclear chromatin contained in the necrotic tissue in which they lie (Unna) it is at times very difficult to demonstrate them in sections. They may be demonstrated in smear-preparations by means of Löffler's alkaline methylene-blue (methylene-blue conc. alcoholic sol. 30 ccm., aqueous solution of caustic potash ($\frac{1}{10000}$) 100 ccm.), the stain acting for 5 minutes, after which the preparation is dipped for 1 second in 1 per cent aqueous solution of acetic acid to which a few drops of an aqueous solution of tropäolin 00 have been added, after which they are quickly washed off with distilled water, dried, and mounted in balsam. Tropäolin decolorises the protoplasm of the cells and their nuclei (partially), but not the bacilli (Löffler). Kühne (1888) advises to transfer sections from alcohol to water, staining with carbolic methylene-blue for 3-4 minutes, decolorising a few seconds with acidulated (HCl) water, washing thoroughly with water, and drying by pressing down upon the section with blotting-paper. The section is then treated 8-10 minutes with aniline oil, to which 20 per cent oil of turpentine has been added, this being followed by oil of turpentine, xylol, and mounting in balsam. Sections thus prepared do not show histological details, and are not durable. Unna (1893) directs to dry the sections upon the slide, stain $\frac{1}{2}$ hour with methylene-blue (according to Löffler, Kühne, Unna); place the slide in water, thus liberating the section, which is then put for some seconds in glycerine-ether mixture, and thence backwards and forwards to water, finally through water, absolute alcohol, oil of turpentine, and mounted in balsam. For double staining he recommends placing the sections in 1 per cent acid fuchsin overnight, washing with water, drying, staining $\frac{1}{4}$ hour in methylene-blue, followed by treatment with 1 per cent arsenious acid for 5-10 seconds, water, alcohol, oil of Bergamot, and mounting in balsam. Abbott (1899) finds the following simpler method useful:—Transfer the sections from alcohol to distilled water, thence to dilute fuchsin kept 15-20 minutes at about 50° C. Transfer to a slide, absorb superfluous stain with blotting-paper, treat $\frac{1}{2}$-$\frac{3}{4}$ minute with 1 per cent acetic acid, remove the acid with distilled water, dry the sections with blotting-paper, wash it 15 seconds with absolute alcohol, clear with xylol, and mount in xylol balsam. Muir and Ritchie (1899) especially recommend carbol-thionin-blue. Add one part of stock-solution, 1 gramme thionin-blue dissolved in 100 ccm. carbolic acid solution (1 : 40) to three parts water, and filter. Stain for 5 minutes or longer, wash thoroughly with water, decolorise with weak acetic acid, wash with water, dehydrate with aniline-oil. The bacilli do not stain by Gram's method. Rosenthal (1888) was able to stain bodies resembling spores in bacilli obtained from old cultures when using Neisser's method for staining endogenous spores. But this is no proof that spores actually occur. Babes (1891) finds that the so-called chromatic portions of the bacilli take on a violet colour with Löffler's stain. Stained with aniline-fuchsin, followed by methylene-blue, the bacilli often show bluish red oval granules within their substance. Marx (1889) finds that the clear spaces observed when staining with carbol-fuchsin may be stained brown by Neisser's stain for *B. diphtheriæ* (methylene-blue and vesuvin), this stain also demonstrating 3-5-9 round dark blue bodies (Babes-Ernst bodies) situated at regular intervals within the bacillus.

Cultivation.—Though pure cultures have been obtained from abscesses they are best made from fresh nodules, as these contain most bacilli. It is best to make the first culture upon potato or blood-serum, as negative results are at times obtained if they are immediately transferred to agar. There are differences observable in the initial cultures which might lead one to suppose that there are different varieties of the bacillus. On potato some rapidly assume their characteristic colour, others do so slowly, whilst some grow well and others badly upon gelatin. Passage through the guinea-pig renders the germ easier to cultivate (v. Babes, 1891). Bacteriologists all agree that the bacillus shows its most characteristic growth upon potato, and, as it grows excellently upon this medium, it is well for purposes of diagnosis to cultivate initial cultures thereon. The bacillus grows best in the presence of oxygen, and at a temperature of 35°-37° C. It grows fairly well at 25°, very slowly at 21°, which may be considered the minimum temperature at which it will multiply. It still grows at 42°, but not at 43° C. It grows better upon *slightly acid or neutral media*, and preserves its virulence longest upon acid potato.

Potatoes are best prepared, according to Kresling (1892), by washing the slices with 0·5-0·7 per cent sodium carbonate solution until the wash-water remains clear. The potatoes are sterilised 80 minutes at 110° C. They should have a degree of acidity corresponding to 0·1-0·3 ccm. of $\frac{1}{10}$ normal soda solution.

Cultures should be prevented from drying. The bacilli will not grow upon potato at room-temperature, being best maintained at 36°-36·5° C. (Marx, 1899). Initial cultures show a brownish, moderately thick, and slimy growth after 3-5 days, at times earlier. Subcultures grow more rapidly, the growth presenting a clear, honey-like appearance, subsequently growing dark and opaque, acquiring a *café au lait* or chocolate colour in the course of a week or so. The potato about the edge of the growth often exhibits a greenish discolorisation. Mashed potatoes spread in a layer upon the bottom of Erlenmeyer flasks also afford an excellent means of cultivating the germ. Next to potato, blood-serum and then glycerinated agar are the best media. On coagulated or gelatinised *serum* after 3 days at 37° small transparent, discrete drop-like colonies appear. Upon *glycerin-agar* slants it forms a somewhat slimy, greyish white translucent growth, which with time acquires a brownish tint. Whereas it grows badly on white of *egg* it grows well upon sterilised yolk, forming knob-like growths after 24 hours (Marx, 1889). It also grows well upon sterilised *carrot*, forming a white pigment in 2-3 days. They form a stringy white mass in gelatin, which takes some weeks to become liquefied. They grow well in glycerinated *bouillon* which has not been neutralised, at first uniformly clouding the medium, and subsequently forming a stringy deposit. Babes (1891) recommends agar made with potato juice instead of meat infusion as a culture medium. Milk is coagulated in 10-12 days at 37° C., the reaction being neutral (Gorini, 1896). It grows in deep gelatin at 22°, and in a hydrogen atmosphere at 37°, but not as well as under aerobic conditions (Marx, 1899).

Products.—We have already referred to the pigment which is formed by the bacillus, and stated that it constitutes an important characteristic of the organism. The pigment varies somewhat, depending upon the nature of the medium. On acid potato it is brown, on agar a pale straw colour, in bouillon at times orange-coloured, on carrots white (Smith, 1889; Marx, 1899). The bacilli exert a pathogenic effect by virtue of toxic substances which they give off or contain. If dead bacilli are inoculated into an animal they produce local pathogenic effects. If the bacilli have first been washed with distilled water they exert less effect. If introduced in sufficient quantity they produce a cachectic condition which ends in death (Babes, Rigler, and Podaska, 1897). Kalning, Preusse, Pearson, Roux, and Foth (1891-96) have by different procedures obtained "mallein" from cultures of the glanders bacillus. Mallein is a compound similar to "tuberculin," and constitutes from a practical point of view the most important product of the germ. Kalning obtained it from potato cultures which he rubbed up with water, heated to 120° C. for 20 minutes on four successive days. He kept the fluid for two days at 39°, and then passed it through a Pasteur filter. Preusse used old dried-up potato-cultures, rubbing them up with glycerine and water, and placed them some days in the thermostat, after which they were filtered and sterilised, the filtrate being the mallein. Roux concentrated the sterilised filtrate of glycerin-bouillon cultures a month old, obtaining a syrupy fluid, which had to be diluted with ten times its volume of $\frac{1}{2}$ per cent carbolic acid for use. The dose of concentrated mallein for a horse amounts to $\frac{1}{4}$ ccm. (Nocard, 1895). The value of mallein in diagnosis has been tested thousands of times upon horses, and in a few cases in man. It is without doubt invaluable in diagnosis, especially of hidden glanders in the horse. Properly prepared it preserves its qualities unimpaired for 1-2 years. Foth (1893-94) has made a dry preparation for which he claims greater stability. (See further under Diagnosis.) De Schweinitz and Kilborne (1894) consider that the active principle of mallein is an albumose which can be precipitated from cultures by alcohol or ammonium sulphate. Its chemical composition has been investigated also by Kresling (1892) and Gutzeit (1892), but our knowledge is still unsatisfactory. Levandowsky (1890) found cultures of the bacillus in bouillon to contain indol and phenol, these according to Kresling (1892) being absent in potato cultures. The bacilli also contain a peptonising ferment, as is seen from the liquefaction of gelatin.

Chemical Composition.—When the bacilli are dried at 110° C. they yield dry substance equal to about 24 per cent of their original weight. The ash (6·67 per cent) contains much phosphoric acid, potassium, sodium, sulphuric acid, and traces of iron and chlorine. The nitrogen amounts to 10·5 per cent. The dry substance is soluble as follows: in water 25·75 per cent, in ether 2·84 per cent, ether and alcohol 3·2 per cent, alcohol 0·66 per cent, the rest remaining insoluble in any of these agents. The ethereal extract yielded a yellow fat melting at 40° C., and containing lecithin, cholestearin, and oleic acid (Kresling, 1892).

Behaviour of the Bacilli outside the Body.—Löffler (1886) did not believe that the bacilli would multiply outside the body under ordinary conditions. They do not do so in hay and other infusions. Bacilli obtained from different sources vary considerably in their powers of resistance. Cadeac and Mallet (1886) found that they could not withstand drying; Bonome (1894) found they died after 10 days at 25°, whilst dried over sulphuric acid they only died after 35 days. Under the most favourable conditions Löffler found that they did not retain their virulence longer than four months. Sanarelli (1889) found them virulent longest (two months) in anaerobic cultures, in aerobic

cultures they lost their virulence in a few days. They have been found to resist putrefaction for 14-30 days, to survive 15-20 days in water. When kept in a moist state at 23° they were still alive and virulent after 20 days, whereas they had died within five days at 15°-16°. They are killed by a $\frac{1}{2000}$ sublimate solution in one hour, by turpentine water ($\frac{1}{100}$) in one hour, by carbolic acid (5 per cent) in five minutes, etc.; in other words, they are readily destroyed by the ordinary antiseptics. When exposed on threads to sunlight they are destroyed in one day. The statements of authors with regard to the effects of heat vary considerably, this being evidently due to the different conditions under which the experiments were carried out. When heated for one minute at 62°, or ten minutes at 55° in culture media, they are killed (Marx, 1899). Bonome (1894), however, found them alive when heated to 70° for six hours in a moist state, though they were killed at 70°-75° in five to six minutes, at 90°-100° in three minutes. Finally Bromberg (1891) found that all the bacilli in old cultures were not destroyed by half an hour's exposure at 100°. (*See also* Sirena and Alessi, 1892; Plemper van Balen, 1897.)

Mode of Infection.—Glanders infection may usually be traced to direct contact with diseased animals, whose discharges from the nose, as also pus, urine, saliva, and milk, have been found to contain the bacilli. The secretions of ulcers and nasal discharge are particularly dangerous. Infection may take place indirectly through harness, blankets, etc., that have been in contact with glandered animals and become soiled with diseased secretions. In many cases a lesion of the skin serves as a port of entry. Babes (1888) considers that infection may occur through the uninjured skin, having succeeded in infecting guinea-pigs by applying a mixture of bacilli in vaseline to their skins. The bacilli penetrated into the hair-follicles, and passed thence through the epithelial layer into the lymphatics. Cornil and Nocard (1890) obtained a similar result in two out of fifteen experiments. V. Babes (1893) cites two cases in man where infection seems to have occurred through the intact skin. It has been claimed, but remains unproved, that infection may occur through blood-sucking flies. Infection has been produced experimentally through inhalation of dried bacilli, and the nasal mucous membranes and lungs may be primarily affected. Lesions in these situations are, however, frequently due to secondary infection, the bacilli having reached the part from primarily affected organs elsewhere. The nasal mucous membrane offers an especially favourable nidus for the multiplication of bacilli carried thither by the blood-current. Friedberger and Fröhner (1896) consider that in nine-tenths of the cases in horses infection occurs through inhalation, but this view is rendered untenable through Nocard's (1894-97) exact experiments proving the readiness with which they may be infected through feeding. Nocard considers the intestinal tract to be the chief port of entry. If pure cultures of the glanders bacilli are mixed with the food or drink of horses, donkeys, rabbits, or guinea-pigs, infection is certain to occur. At autopsy numerous minute translucent or even calcified nodules are observable in the lungs. The negative results obtained in such experiments by Cadeac and Malet (1894) may be explained through their using less virulent cultures than did Nocard. Infection through feeding has certainly occurred spontaneously in dogs, cats, and animals in menageries. Infection of the fœtus occurs in a limited number of cases in the horse, dog, and guinea-pig (Löffler 1886, Cadeac and Malet 1886, observed this twice in thirteen experiments; Bonome 1894). A few cases of infection have occurred in bacteriological laboratories, once through the prick of a hypodermic needle whilst inoculating an animal. Persons who have to do with horses are naturally most affected, nevertheless some cases have been observed where no such connection could be traced. Zawadzki, and Nencki and Pruszynki (1896) report the case of a physician who infected himself whilst operating upon a fellow-practitioner who also subsequently died of glanders. Veterinarians have become infected through being bitten whilst examining a horse's mouth, or through secretion being thrown upon the apparently intact conjunctiva. Rémy (1897) has collected nine cases of man to man infection. He cites the case of a woman who became infected whilst making horse-hair mattresses, as also that of a person who acquired glanders from being struck by the fist of a man who owned a glandered horse. Grawleswki (1893) cites the case of a washerwoman who developed a glanders pustule three days after washing the clothes of a man who had died of the disease. He also tells of an old man who attended a person who died of glanders, and developed initial lesions in places where he was accustomed to scratch himself. Tessier (1852) and Cooper (1887) made observations which indicated that infection may occur in man through coitus. Gibert (1840) and others since have proved the auto-inoculability of glanders in man and animals. It is evident, then, that a man may infect himself in other parts of his body through scratching himself.

Increase and Decrease of Virulence.—The bacillus rapidly loses its virulence in cultures, so that it is necessary from time to time to pass it through a susceptible animal. Gamaleia (1890) was able to increase the virulence by continued passage through Spermophilus, and Foth (1896) did this with field-mice. Tedeschi (1893) did this through intracerebral inoculation of susceptible animals, being able to kill

dogs with the more virulent bacilli. Sacharow (1893) raised the virulence for cats by repeated passage (eight) through this species, but found then that the bacilli had lost their virulence for horses, a fact which has been observed in other germs. Straus (1889) claimed that they became attenuated by passage through the wolf. It is certain that the bacilli vary greatly in virulence even when taken directly from the horse (Kitt, 1896), and tested upon horses and guinea-pigs.

Experience in Russia, as also observations reported by Semmer (1894) in Germany, prove the existence of a mild form of glanders. This milder form is often referred to as "southern glanders," because of its greater frequency in France, Italy, and Southern Russia. It seems to be unknown in Northern Russia, Germany, and Scandinavia. Whether this depends upon a virus of lessened virulence or upon an inherited resistance of certain southern and eastern races of horses is not determined.

VI. Immunity.—We have noted above the immunity or susceptibility shown by certain species of animals towards this disease. There exists also an individual immunity or susceptibility. It has, for instance, been repeatedly observed that certain horses could be exposed with impunity for months or even years by being kept in stables with glandered animals. This natural resistance may, however, be overcome through overwork, bad food, neglect, poor stabling, exposure to cold or the debilitating effects of intercurrent disease. This explains the greater prevalence of glanders amongst horses in times of war. One attack of glanders seems to convey a certain degree of immunity, but the evidence can scarcely be termed satisfactory. Experiments at producing immunity through inoculation with dead or attenuated cultures, as also with the serum of naturally immune animals, or such as have recovered from an attack, or been rendered resistant to increasing doses of the virus, as also inoculations with mallein, have not given fully satisfactory results. Some investigators report positive results, others negative ones, others again that they have obtained inconstant results. (Straus 1888, Charrin 1885, Finger 1889, Sadowsky 1891, Chenot and Pick, Semmer 1892, Sacharow 1893, Semmer 1894, Hüppe, Schindelka, Bonome, Schattenfroh 1894, Preusse, Nocard, Comeny, v. Babes, Rigler, and Podaska 1897.)

Since masked glanders has been more often detected through the use of mallein in diagnosis, it has been made quite clear that glanders is by no means the fatal disease it used to be thought. Faverot and Humbert (1893) state that as long as the nodules are grey and transparent it is possible for the animal to overcome the bacilli; when the nodules undergo cheesy degeneration the bacilli usually overcome the animal. Animals kept under good hygienic conditions stand a much better chance of recovery. As this was determined upon animals but slightly affected, mallein being used for diagnosis, it does not alter the prevailing opinion based upon well-marked clinical appearances, for the more developed disease is practically incurable. Nocard (1896, 1897) holds strongly to the opinion that recovery may result in quite fresh cases, for the reason that he has frequently observed spontaneous recovery under ordinary conditions as also in experimentally infected animals. Rémy (1897), who has gone over the literature, cites twenty-three recorded cases of recovery from glanders in man between the years 1812-92. Whilst acute glanders or farcy may lead to a fatal termination in 8-15 days, the chronic form may last for months or even years. In chronic cases death may be caused by pyæmia or acute glanders. Hallipeau and Jeanselme report the case of a man who suffered six years. The disease was characterised during the first three years by recurring phlegmons and abscesses, but no general symptoms, the lesions healing spontaneously, or as the result of treatment with caustics. A latent period of three years now followed, when the disease broke out again in the acute form, the man dying. Bollinger observed a case of eleven years' standing.

M'Fadyean (1896), and subsequently Foulerton, Wladimiroff, Bourges and Méry (1897-98), observed that the serum of a glandered animal agglutinated bouillon cultures of the glanders bacillus. Semmer (1892) was unable to produce immunity in cats and guinea-pigs through the serum of naturally immune animals such as the horse and cattle, whilst Chenot and Pick (1892) claimed they had been able to save seven-tenths of infected guinea-pigs by the use of cattle serum. When guinea-pigs had been inoculated with very virulent cultures (which killed in five days) the serum retarded death until the 21-42 day. V. Babes, Rigler, and Podaska (1897) state that donkeys treated with increasing doses of mallein, and finally with injections of dead bacilli, yield a preventive and curative serum. Semmer treated horses with mallein and found their serum only conferred a transitory immunity.

VII. Symptoms.—*In the horse* acute glanders has a period of incubation varying from 3-8 days or longer. At the onset febrile symptoms manifest themselves, the horse loses its appetite and is sluggish. In a day or two a serous nasal discharge appears, which at first may be from one nostril only; by the fifth or sixth day this discharge is purulent, yellow, and viscid. The margins of the nostrils are swollen and the lymphatics of the face inflamed. The nasal mucous membrane, at first hyperæmic, inflamed, and studded with small nodules, later shows large irregular ulcerating patches, which tend to become confluent and form large serpiginous ulcers with raised edges. These ulcers may eat

into the deeper tissues, and lay bare and erode the cartilage of the septum and the nasal bones. The submaxillary lymphatic glands are enlarged, indurated, and in some cases become adherent to the surrounding tissue; they are not tender and seldom ulcerate. Hot, painful œdematous swellings appear on the trunk and limbs; if on the latter they cause lameness. The disease reaches its height in from 6 to 15 days, the animal being by this time in great distress. Breathing is snuffling and difficult, the animal constantly snorting to expel the mucus and viscid nasal discharge which glues together the alæ nasi. The lymphatics of the face and other parts of the body are often inflamed and corded (acute farcy), and death from suffocation preceded by painful dyspnœa follows rapidly.

Acute Cutaneous Glanders: Farcy.—The onset resembles that of acute glanders, and as a rule acute glanders is present at the same time; if not, it usually supervenes before death. The characteristic local lesions are inflammation of the lymphatic vessels, which appear as firm cords running under the skin, and the formation of nodules situated at intervals along these cords. The nodules show a marked tendency to ulcerate and discharge a thick oily material. These changes may appear in any part of the cutaneous surface, but show a preference for the hind limbs. The ulcers form about the sixth day. They have irregular raised borders, with a yellowish base covered with a grumous discharge. Death follows rapidly, usually from acute glanders, involving the respiratory tract.

Chronic Glanders.—This form of the disease often comes on insidiously; there is frequently some constitutional disturbance, which may subside as the local lesions are developed. The premonitory symptoms vary much. The general health may appear good, but there is some dulness of the eye and roughness of the coat, the animal shows lack of endurance and sweats easily. Lameness, intermittent or continuous, may be noticed, there may be œdema of one or more limbs, cough, bleeding from the nose, and possibly orchitis. The nasal discharge often present is at first clear, but eventually becomes cloudy, purulent, and viscid, and the animal is continually snorting to expel the secretion. If the nasal mucous membrane is examined, nodules and ulcers can usually be seen, and after the disease has existed some time stellate scars are also present. The submaxillary lymphatic glands may be enlarged, but as a rule do not ulcerate. The larynx and trachea may be the seat of the primary lesion, the nasal symptoms being delayed. In these cases, cough with varying expectoration, enlarged glands, tender larynx, fœtid breath, and general ill-condition are usually present. The skin (chronic farcy) may be attacked before the respiratory passages. Nodules form and ulcerate, discharging a thick oily material, and generally after a few weeks heal, leaving a dense stellate cicatrix, fresh nodules forming in adjacent or more distant parts, these again healing, to be followed by the formation of fresh nodules. The lymphatics leading from the affected parts are corded (farcy pipes). This process may go on for months or years, the animal either recovering or dying from an acute attack.

Symptoms in Man.—The period of incubation is from 3 to 8 days, but apparently it may extend to three weeks. The disease commences with malaise, headache, vague pains in the muscles and joints simulating rheumatism. If the bacillus has entered through a wound, this may completely heal, but in a day or two the seat of inoculation becomes painful and swollen, an eruption of vesicles often appears around it, and the lymphatics become swollen and painful. Frequently the skin about the seat of inoculation resembles phlegmonous erysipelas. In other cases gastro-intestinal symptoms are most prominent, and the disease simulates typhoid fever. Occasionally a pustular eruption, not unlike that of small-pox, occurs on the face and limbs. In most cases the general symptoms rapidly increase in severity, the joint pains becoming more severe, and œdematous swellings appear on the limbs. The temperature is raised, and though at first the rise may be intermittent, it soon becomes continuous. It is accompanied by severe headache, great prostration, dry skin, foul mouth, and fœtid breath. The pulse is full and soft, varying between 90 and 100. Epistaxis often occurs. Hard, painful nodules may appear in the skin; these soon soften, break down, and form extensive, deep, irregular ulcers which may even lay bare the bone. The affections of the mucous membrane of the nose appear to be less frequent in man than in the horse, but they do occur. There is at first a feeling of dryness of the mucous membrane, accompanied by pain and tension at the root of the nose. The skin over the bridge of the nose becomes swollen and reddened, the mucous membrane is much injected and swollen, a fœtid secretion appears, at first clear and thin, but soon becoming yellow, purulent, and viscid, and finally blood-stained. The eyelids and conjunctivæ are swollen and œdematous, often completely closing the eye. The conjunctiva secretes a purulent fluid. The inflammatory process is apt to spread, invading the pharynx and palate. The gums are swollen and bleed readily, the breath is fœtid, swallowing is difficult, and the voice hoarse. The lungs are also involved, severe pleuritic pains are complained of, and there is a troublesome cough. Towards the end œdema of the glottis may supervene. The bowels, at first obstinate, become relaxed, and profuse diarrhœa follows. If the liver is involved it is enlarged, painful on pressure, and there may be jaundice. As the disease progresses sleeplessness and nocturnal delirium ensue. If

nodules form in the muscles, rigors simulating those of pyæmia are a prominent symptom. Towards the end the heart fails, its beats become quickened, the pulse becomes thready, easily compressible, the temperature rises to 105°-106° F., the skin is covered with a cold clammy sweat, consciousness becomes cloudy, the evacuations are passed involuntarily, and eventually the patient becomes comatose. The extremities are cold, gangrene and ulcers extend, giving rise to a fearful stench, and finally, after complete loss of consciousness and sensibility, dyspnœa increases, the breathing becomes stertorous, and tetanic convulsions close the scene.

Chronic Glanders.—This form of the disease may last months or even years; the patient complains of a vague feeling of discomfort, pains in the extremities and joints, loss of appetite, and sleeplessness. The attacks of fever are recurrent. The symptoms vary in severity from time to time, and eventually either pass into an acute form, or complete recovery may take place. The local lesions in these cases vary greatly. If the skin is the seat of the lesion the surrounding parts become swollen and œdematous, the nodules usually appear in one part only; they break down, leaving indolent ulcers, which may heal, leaving a scar. Abscesses form around joints, and fistulous openings, which show little tendency to heal, appear upon the surface. They discharge a thin foul pus. An eruption of papules, which become pustular, may appear. Affection of the nasal septum is often absent, when it appears it follows a similar course to that met with in the acute disease, but is much more chronic. Extensive ulceration occurs, leading to destruction of the septum and even gangrene of the root of the nose. Inflammation and ulceration of the pharynx and soft palate may occur, and in these cases the lesions strongly resemble those found in syphilis.

VIII. Pathology.—The lesions which occur in this disease are due to the *B. mallei* and its products. This organism lodging in the tissues gives rise to a series of changes, the most characteristic being the formation of proliferative nodules in various situations, chiefly in the mucous membrane of the respiratory tract, lungs, and skin. But they are also found in the subcutaneous and submucous tissue, liver, kidney, dura mater, periosteum, and synovial membranes. These nodules show a marked tendency to break down, forming ulcers if situated on free surfaces.

In the horse a common situation for these lesions is the nasal mucous membrane. Within two or three days of infection a small injected area appears, in which a yellow nodule is seen projecting from the free surface. This yellow centre breaks down and leaves a small ulcer. The nodules usually occur in groups, and the ulcers running into each other may soon form a large ulcer, with irregular indurated edges, a deep infiltrating base. This is surrounded by many small nodules in various stages of development and softening, which, breaking down, increase the area of the ulcerated surface. The ulcers at first discharge a thin glairy fluid; this rapidly becomes purulent and viscid. In addition to these changes the mucous membrane is often swollen and injected, and secretes a viscid mucus which appears to possess specially irritating qualities, leading to superficial ulcerations of the mucous membranes in situations where no nodules are found. The ulceration, very extensive and destructive, may lead to perforation of the septum and necrosis of the nasal bones. Characteristic nodules, at first translucent, hard and shotty to the touch, form throughout the lungs. These are surrounded by an area of consolidated lung which microscopically shows a proliferation and infiltration of the walls of the air vesicles and exudation of leucocytes, with the formation of fibrin in the air vesicles themselves. The area is usually surrounded by a zone of injection, which is especially well seen on the pleural surface. In chronic cases the nodules become fibrous and even calcified. In acute cases the submaxillary lymphatic glands are enlarged and indurated, and tend to become adherent to the surrounding structures. In the skin the lesions usually run a more chronic course; nodules, diffuse swellings, and inflammation and enlargement of the lymphatics leading from the part occur. The nodules break down and form ulcers and sinuses, which show but slight tendency to heal. In the more chronic cases the ulcers both on the nasal septum and in the skin may heal, leaving dense stellate scars. Hot, painful, œdematous swellings are frequently seen.

In man the process is usually very acute. If the septum nasi is involved, the formation of nodules occurs as in the horse, but the ulceration in this case is usually more rapid, and the destruction of the deeper tissues more extensive; perforation of the septum and necrosis of the nasal bones often occurring within a few days of onset. Ulcers also form on the soft palate and pharynx. The antrum and sphenoidal sinuses may be also involved, and occasionally the inflammation spreads into the cranium, causing inflammation of the meninges. If the lungs are attacked numerous nodules are found scattered throughout their substance. These are small, and surrounded by an area of consolidated lung tissue with a bright zone of injection. If cutaneous lesions are present they commonly occur as papules, which rapidly become pustules, containing a cheesy material or thick, blood-stained pus. A red scab forms, which, if removed, leaves a small ulcer. Œdematous swellings also occur, and in many cases show a striking resemblance to phlegmonous erysipelas. The muscles may contain nodules

which caseate; these also frequently occur in the periosteum, especially at the insertion of tendons. In acute cases the spleen is usually enlarged and soft. The liver shows cloudy swelling, and may contain nodules scattered throughout its substance. Similar changes are described in the kidney. Secondary infection may occur, more especially in chronic cases.

IX. Diagnosis.—The diagnosis will depend upon the clinical appearances already referred to and the finding of the specific bacillus, the positive reaction with mallein and the Straus test.

Straus Test.—Dilute the pus, secretion, or juice of diseased gland in sterile water or bouillon, and inject the fluid respectively into the peritoneal cavity and subcutaneously into male guinea-pigs. If impure material is injected (nasal discharge) it is inadvisable to trust to one guinea-pig, as it may die of suppurative peritonitis before the characteristic orchitis has had time to develop. Where the guinea-pig is inoculated with pus containing few bacilli the orchitis may be late in appearing. Levy and Steinmetz (1895) advise to make the intraperitoneal injection in the median line above the bladder, so as to avoid injuring the naturally large testes. Two to three days after injection the testes swell, the scrotum becomes reddened or purplish and glossy, adhering to the subjacent tissue, it being impossible to return the testes into the abdomen. The animal dies in 4-8-15 days. At autopsy one finds a violent inflammation of the tunica vaginalis, the serous membrane being covered by yellowish white granulations the size of a pin's head, the opposing surfaces being glued together by thick purulent exudate containing many glanders bacilli. The testicle and epididymis are very rarely affected. Usually this orchitis is sufficiently developed by the third or fourth day to make a diagnosis possible. The value of this test has been tried very generally (Straus, 1889; Hallipeau and Jeanselme, Finkelstein, Levy and Steinmetz, Kitt, and especially Nocard, 1891-96). At first its value was overrated. As Kitt pointed out, some guinea-pigs show a marked resistance, which takes away from the value of a negative result. The orchitis may be produced by other germs, *B. orchiticus* (Kutscher, 1896), which has the same morphological characters as *B. mallei*, but is distinguished therefrom by its retaining the Gram stain and behaving differently in cultures. The bacillus of epizootic lymphangitis (Nocard, 1892-96) is also different from *B. mallei* in these respects, consequently the staining test must also be applied to the organisms causing the orchitis. Secretion may be obtained from the nasal cavity by means of a sterile cotton swab, which is afterwards rinsed in sterile fluid for injection.

Mallein Test.—When mallein is injected into a glandered horse a typical reaction follows. After some hours an inflammatory swelling appears at the seat of inoculation; it becomes hot, tense, very painful, and always extensive, at times enormous. From the borders of the swelling hot and painful sinuous lymphatic swellings radiate toward the neighbouring glands. Suppuration never occurs where the inoculation has been carried out aseptically. The tumour increases in size for 24-36 hours, persists several days, subsides slowly, and disappears in 8-10 days. With the appearance of the swelling a general reaction takes place, the animal appears dejected, anxious, the coat ruffled and dull, breathing is accelerated, the appetite lessened. The animal shivers or has at times violent convulsive attacks. This constitutes the "reaction organique" of Nocard (1896), which is invariably accompanied by the febrile reaction which appears within 8 hours after the injection, and reaches its maximum in 10-12 or at times 15-18 hours. The rise of temperature amounts to 1·5°-2°-2·5° C., and persists for 24-48 hours. In healthy animals the injection of even much larger doses has little or no effect; a small, hot, œdematous and sensitive swelling may appear at the seat of inoculation, but it disappears entirely in less than 24 hours. A reaction has been observed in horses suffering from melanosis, bronchiectasis, and pulmonary emphysema, but it is not typical, and should be checked by the result of the Straus test. Where a doubtful reaction occurs the injection should be repeated after the temperature has returned to normal. To guard against error, animals should not be injected when showing fever, and they should be kept in the stable at a uniform temperature (changes in outside temperature have been observed to cause a variation of 1·5°-2° C. in the rectal temperature). (Kalning, Preusse, Pierson, Babes, Eber, Heyne, Schilling, Peters and Felisch, Dieckerhoff and Lothes, Bang, Nocard, Foth, Guinard, Teissier, Rabieux, Cadiot and Roger, Nikolsky, Schindelka, Johne, M'Fadyean, Remmert, Semmer, etc. etc., 1891-97).

Bonome (1894) tried the effect of mallein on a man suffering from chronic glanders of the nasal mucous membranes and cervical glands. A violent general reaction followed, the temperature rising after 4-7 hours. The mucous membrane of the nose and eyes became swollen, the pulse accelerated, the urine increased in amount. At first the reaction was proportionate to the amount of mallein used, but grew less with repeated doses; the patient, he says, showed marked improvement after two months' continued treatment.

X. Treatment.—The treatment of this disease is most unsatisfactory, and must be mainly symptomatic. If the case is diagnosed early, and an infective lesion found, it should be removed by the knife. The ulcers may be treated with antiseptics, and the fœtid smell kept within bounds by some deodorizer. In chronic cases

fresh air, light, and good food will do more than any drug. Mallein has been claimed to be beneficial in the treatment of horses.

XI. Prevention.—As soon as it has been determined that glanders exists in a stable, all affected animals should be killed, and others which have been exposed should be isolated until they have been tested with mallein. The slaughtered animals should not be skinned, but put out of harm's way as soon as possible. Where the bodies cannot be rendered innocuous by boiling, destruction with chemicals, or fire, they should be buried at a spot some distance from the stable or house. The grave should be deep enough to allow of the cadaver being covered by a layer of soil at least 3 feet deep. As soon as a positive diagnosis has been arrived at it should, if possible, be determined how long the symptoms have existed, and if animals from other parts have been exposed to the infection. Where this is the case the animals should be inspected and watched. Persons who have to do with glandered or suspected animals should be thoroughly warned regarding the danger to which they are exposed. The infected stable should be closed to other horses and to every person outside of those directly concerned. Stablemen attending diseased animals should keep away from healthy ones. Suspicious animals should be examined every two weeks by a competent veterinarian, and though they may be worked as long as they show no symptoms, they should be kept apart from the unexposed, and watched for 3-6 months. The harness, etc., used by such animals should be reserved for their use alone. Where glanders has existed, the stables, harness, blankets, saddles, waggon-poles or shafts, buckets, drinking-troughs, mangers, etc., should be thoroughly disinfected. The stables should be cleaned out and aired, the walls and wood-work white-washed. Sulphur may be burned (20 grams per cubic meter space) or chlorine evolved (add 10 grams of hydrochloric acid to 5 grams fresh chloride of lime for each cubic meter), and allowed to act for 8 hours, all apertures in the stable being sealed. Sulphur should not be used when chloride of lime is applied. Where the floor is bad, and the ground under it liable to have become soiled with the discharges of sick animals, the floor should be removed, as also the surface soil beneath, lime being freely scattered upon the spot. Where floors are good they may be washed with milk of lime or carbolic acid solution. Bits, chains, and metal articles may be sterilised over a fire, or scrubbed with boiling water and carbolic acid solution; articles having no especial value should be burned. Harness and leather articles may be washed with hot soda solution (50 grams soda to 10 litres water) or with hot soap-suds, being afterwards dried and rubbed with carbolised oil. Saddles, etc., which will not bear such treatment may be exposed to sulphur or formalin vapours. From this and what has been said with regard to the mode of spread of the disease, other precautionary measures will suggest themselves in particular cases. Naturally personal measures are indicated for those who are exposed, the hands, shoes, or soiled clothing being disinfected with carbolic acid, etc., when soiled. The possibility of a glandered horse being used for the production of an anti-diphtheritic serum has been suggested as a source of danger to man, but Bonhoff (1897) finds that the 0·5 per cent carbolic acid added to such serum kills off the bacilli in 2-4 hours.

Glands. *See* Bronchi, Bronchial Glands; Lymphatic System; Physiology, Blood (*Hæmolytic Organs*); etc. etc.

Glandulæ Concatenatæ. *See* Syphilis (*Secondary*).

Glandulæ Suprarenales. *See* Adrenal Glands.

Glandulæ Thyroideæ. *See* Thyroid Gland (*Anatomy and Physiology*).

Glandular Fever.

Glandular fever is an acute infectious fever characterised by inflammation, apparently primary, of lymphatic glands, beginning in the deep cervical group and sometimes limited to it, and by constipation. The epidemic nature of the disease was first maintained by Pfeiffer in 1889, and German writers frequently refer to it as Pfeiffer's "Drüsenfieber."

The disease attacks children especially, and when introduced into a household few between the ages of six months and thirteen years escape; adults, though not altogether immune, appear to contract the disease much less readily.

The incubation period varies from five to ten days, but the most usual period is seven days.

The onset is usually quite sudden, the first complaint being of pain in the neck and headache. The pain is aggravated by movement and the head is held stiffly. It will now be found that the temperature is raised (101° to 103° F.), that the face is flushed, the tongue furred, and that there is deep-seated tenderness in the neck, especially in the anterior triangle, though no enlargement of glands may be perceptible. In many cases abdominal pain is an early symptom, and there may be tenderness on deep pressure. Enlargement of the liver can be made out in most cases, and of the spleen in some. There is no jaundice. The child refuses food, and suffers from nausea or

vomiting. There is usually some pain on swallowing, but there is either no acute pharyngitis, and this is the rule, or only some slight catarrh which is insufficient to account for the dysphagia and pain in the neck. Constipation is nearly always present at this stage, but in very mild cases small mucous stools may be passed at frequent intervals. After these symptoms have persisted for two or at most three days, attention is drawn to an oval swelling beneath the sterno-mastoid muscle, and presenting at its anterior border. In almost all cases this swelling is situated on the left side, and is perceived on palpation to be due to two, three, or more glands. Medical aid is often first sought at this stage. The enlargement of the glands continues to increase for two or three days more. At the end of this period the pain in them ceases, and they decrease in size. Meanwhile adenitis has probably begun in other groups of glands, and runs a similar course. The glands most usually affected second in order are the corresponding group on the right side, but the posterior cervical, the axillary, the inguinal, and the mesenteric may become enlarged and tender, and some observers believe that the last-named are involved in at least half the cases. The temperature commonly attains its maximum, it may be as high as 104° F., on the third day, when the adenitis of the first group of glands to be involved is in its most acute stage. Its later course is uncertain, and depends apparently on the number of groups of glands attacked. There is no distinct defervescence so long as fresh groups are being attacked. Convalescence usually begins at the end of a fortnight. The commencement of defervescence is frequently marked in severe cases by the passage of numerous copious green stools containing much mucus. The pulse continues rapid for a day or two after the temperature falls. The enlargement of the glands diminishes slowly, but eventually it disappears completely. Suppuration seldom or never occurs. The severity of the disease varies very much in different cases. As a rule it is a mild affection, but occasionally the symptoms are severe, and suggest the onset of typhoid fever. Such cases may be complicated by nephritis, and there may be blood in the urine. Convalescence even after mild attacks is generally prolonged. The child is left in a condition of pronounced anæmia, it is languid and disinclined to exertion, appetite is poor, and even under favourable circumstances it may be several months before the health is thoroughly restored.

The diagnosis, as will have been gathered from the above description of the symptoms, must be made mainly by exclusion. The symptoms are such as will call attention to the probability of acute disease of the tonsils or pharynx. The sudden onset, after a day or two of fever, of stiffness of the neck, followed in a couple of days or so by enlargement of lymphatic glands on one side of the neck, is, especially if accompanied by abdominal pain and constipation, very suggestive of glandular fever. Suspicion will be confirmed by the occurrence of other similar cases among children resident in the same house, and it should be remembered that in the same family some cases may be so mild as easily to escape attention, while others may be so severe as to raise a suspicion of typhoid fever.

The pathology of the disease is obscure, and will probably not be cleared up until opportunity has been found for a thorough bacteriological study of an epidemic. It is possible, if not probable, that the primary departure from health is some infection of the intestine; the enlargement of the mesenteric glands observed in a large proportion of cases is held to be secondary to this, and it has been sought to account for the fact that the cervical glands are affected first on the left side by the assumption that the infection is conveyed by the thoracic duct. The resemblance which the disease presents to the mildest form of bubonic plague (pestis minor) and to the "non-venereal bubo" of military writers will not escape attention.

Treatment must be directed to the relief of symptoms, for there appears to be no means of modifying the course of the disease. Thus hot fomentations or belladonna applications will relieve the pain attending the enlargement of the glands which usually commences on the left side of the neck, but will not prevent the subsequent enlargement of those on the right side. Constipation may be relieved by laxatives, or by a dose of calomel, but it soon becomes re-established, and repeated purgation is inadvisable. Some advantage may perhaps be derived from the continuous exhibition of small doses of calomel (gr. $\frac{1}{10}$ to $\frac{1}{3}$ or $\frac{1}{2}$) three or four times in the twenty-four hours, and antipyrin is of use to relieve the headache. Sodium salicylate is probably the most useful drug in the early stage when the temperature is high and the pain and stiffness in the neck severe. While the temperature is raised, the child should be kept in bed and receive only fluid nourishment. After convalescence has commenced, the patient should have as much fresh air as possible, but should be warmly clad, especially about the abdomen. Iron and tonics are indicated, and change of air is often necessary to obtain complete restoration to health.

Glans Clitoridis. *See* GENERATION, FEMALE ORGANS OF (*External*).

Glans Penis. *See* PENIS, SURGICAL AFFECTIONS OF.

Glass-Blowers. *See* LUNGS, EMPHYSEMA OF (*Inducing Causes*).

Glass-Rod Test.

See Ocular Muscles, Affections of (*Abnormal Position, Tests*); Strabismus (*Detection*).

Glass-Workers.

See Trades, Dangerous (*Plumbism from Glass-cutting*).

Glaucoma.

See also Amblyopia (*Diagnosis*); Cataract (*After Treatment of Cataract Extraction*); Choroid, Diseases of (*Sarcoma, Diagnosis*); Cornea (*Ulcerative Keratitis, Causes*); Gout (*Irregular, Eye Conditions*); Headache (*Diagnosis of Cause*); Sclerotic, Diseases of (*Staphyloma, Infantile Glaucoma*); Syphilis (*Secondary, Eye Symptoms*); Vision, Field of (*Field in Glaucoma*).

Definition and Nature.—The disorder of the eye known as glaucoma depends essentially on an excess of pressure in the chambers. In the healthy eye the fluid which fills the chambers keeps the tunics in a state of moderate tension, and thus preserves the form of the globe. Its pressure is equal to about 25 mm. of mercury; it varies somewhat with the force of the circulation, but only within narrow limits. Under certain circumstances the balance between the secretion and the excretion of this fluid is disturbed, an accumulation occurs in one or other chamber, the pressure rises, the tunics become more tense, the circulation of blood in the choroid and retina is embarassed, and the function of the eye is seriously disturbed. This is the condition which we call glaucoma. The name referred originally to a greenish discoloration of the pupil, but this, though sometimes seen in the later stages, is no necessary part of the disorder. The name as now used denotes the peculiar morbid process which depends on pressure.

When glaucoma arises in an eye which appears to be otherwise healthy, we call it *primary*; when it occurs as a complication of some other disorder of the eye, *secondary*. Every eye which is suffering from an excess of internal pressure, however caused, is in a glaucomatous condition.

Causes and Pathology.—The ciliary processes secrete a fluid which nourishes the vitreous body and lens, and fills the aqueous chamber. A very small part of this fluid passes slowly backward through the vitreous and leaves the eye in the region of the optic nerve; by far the larger portion passes forwards through the pupil and leaves the eye at the angle of the anterior chamber by filtering into Schlemm's canal and the minute veins connected with it. Glaucoma appears to depend in all cases upon some change which retards or prevents the escape of the intraocular fluid. In the large majority of cases the filtration angle is compressed or closed; in a few it remains widely open, but its permeability is diminished by inflammatory changes in the filtration area, or by blocking with blood, albuminous exudations, lens matter, or other abnormal contents of the aqueous chamber. These changes arise in various ways.

Causes of Primary Glaucoma.—In persons whose eyes are predisposed to the disease, an acute attack may be brought on by exposure to cold and damp, by fatigue, hunger, loss of sleep, or excessive mental effort, by anxiety or depressing emotion, by constipation, vomiting, or alcoholic excess, in short by any condition which disturbs the cerebral circulation and causes congestion of the eyes. Sometimes an attack begins during the course of a febrile disorder, such as pneumonia or influenza, or it may arise through a local cause, such as an injury of the head, a contusion of the eye without apparent lesion, or a slight abrasion or burn of the cornea. In certain eyes, where the anterior chamber is shallow and the filtration angle already narrow, the use of atropine or any other dilator of the pupil may light up a severe glaucoma, by thickening the base of the iris and thereby causing a complete blocking of the filtration angle; it is chiefly in the eyes of elderly people that this disaster is to be feared.

If we examine an eye which has been recently

Fig. 1.—From the healthy emmetropic eye of a man aged 57. For comparison with Figs. 2 and 3.

blinded by acute glaucoma, we find the lens and ciliary processes pressed forward by an accumulation of fluid in the vitreous chamber; the pro-

cesses much swollen and sometimes greatly altered in shape by pressure against the lens; and the base of the iris firmly pressed against the periphery of the cornea by the forward pressure of the processes. If the eye have been already blind for some months, as a result of acute glaucoma, the base of the iris will be found firmly adherent to the periphery of the cornea. At a later stage still the ciliary processes, instead of being enlarged and pressing forwards, will be found atrophied and retracted (see Figs. 1, 2, and 3). Swelling of the ciliary processes through venous congestion is probably in many cases the starting-point of the attack. In others there is probably some initial change in the zonula or hyaloid membrane, in the substance of the vitreous, or in the vitreous fluid itself, which prevents the overflow of this fluid from the vitreous into the aqueous chamber, and thereby leads to overfilling of the vitreous chamber. In other cases, again, it is probable that a forward displacement of the lens arises through loss of tension in the zonula. However it may be brought about, the compression of the filtration angle raises the pressure in the whole eyeball; this impedes the flow of blood through the choroidal veins and adds to the congestion and swelling of the processes; and this in turn aggravates the compression of the filtration angle. Acute glaucoma, like strangulated hernia, presents an obstruction of the circulation which intensifies itself, and which can only be relieved by removing the injurious pressure. In the slow chronic form of primary glaucoma there is little sign of vascular congestion, and there is no sudden or complete closure of the filtration angle; in the early stages the hindrance to the escape of the aqueous fluid is comparatively slight.

FIG. 2.—Primary glaucoma, acute, recent. Ciliary processes swollen and advanced; iris-base pressed against cornea, but not adherent to it.

The predisposition to primary glaucoma appears to depend largely on certain structural changes or peculiarities in the eye which bring the lens into closer relations than usual with the parts around it. The crystalline lens, so long as it remains healthy, continues to increase in size throughout the whole of life, whereas the globe is fully grown at the beginning of adult life, if not earlier. Hence, as age advances, the lens encroaches more and more on the space in which it lies, its margin coming into closer relation with the ciliary processes, and its anterior surface approaching the cornea. In some eyes these changes pass the limit of safety; the ciliary processes, having insufficient free space at their disposal, are apt, especially during periods of turgescence, to press against the iris-base, and thus compress the filtration angle, and the iris being already very near to the cornea, tends, when the pupil is dilated, to block the outlet. In accordance with these natural changes in the eye, the liability to primary glaucoma steadily increases throughout life; it is extremely slight in childhood and early adult life; it increases in each decade; it is manifest chiefly in elderly people; at sixty-five years of age, for example, the liability to the disease is at least twice as great as at forty-five. The liability of women is greater than that of men in a ratio probably of about six to five, and in women the disease more often occurs in the acute congestive form.

Again, in eyes of small size the relations of the lens are often faulty. The growth of the lens appears to be little influenced by variations in the size of the parts around it, and in eyes in which the cornea and sclera have failed to reach their full development the lens is apt to be relatively too large. This is very obvious in certain cases of microphthalmos. A small eye may usually be recognised during life by smallness of the cornea. The horizontal diameter of the cornea measures normally from 11 to 12 mm.; among eyes in which it measures less than 11 mm. we find a higher percentage of glaucoma, and those in which it measures as little as 10 mm. seem seldom to escape the disease.

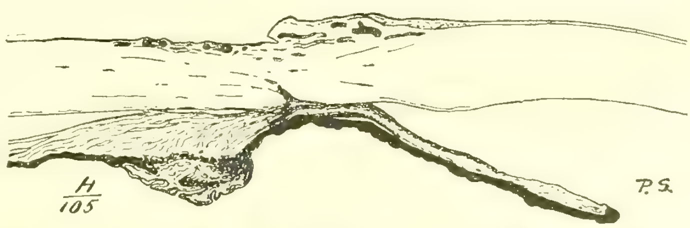


FIG. 3.—Primary glaucoma; chronic; duration about 12 months. Iris-base adherent to cornea; ciliary processes atrophied.

The predisposition to glaucoma is sometimes distinctly hereditary. In one such case observed by the writer both parent and child had exceptionally small eyes.

Certain races—Jews, Egyptians, negroes—are said to be specially liable to the disease, but on this point more precise evidence is wanting. Among Egyptians the average cornea is said to be smaller than among Europeans, and smaller

in eyes suffering from primary glaucoma than in healthy eyes.

Hypermetropia probably predisposes somewhat to glaucoma in advanced life, for the hypermetropic eye has as a rule a more prominent ciliary body and perhaps a shallower anterior chamber than the emmetropic or myopic eye, and these conditions tend to facilitate compression of the filtration angle. But the influence of hypermetropia in this respect has hardly yet been proved by statistics; it must be remembered that the acquired hypermetropia produced by changes in the lens in advanced life is a very common condition at the age when glaucoma most commonly begins, and it must not be assumed that small eyes are necessarily hypermetropic, for this is not the case. It is the small eye as such rather than the hypermetropic eye that is peculiarly liable to primary glaucoma.

Apart from these physiological variations it is probable that degenerative changes in the blood-vessels and other tissues of the eye account in part for the predisposition to primary glaucoma which belongs to advanced life.

Causes of Secondary Glaucoma.—Sarcoma of the choroid usually leads to glaucoma unless the eye be excised before the outbreak. The glaucoma is of acute type and closely resembles the primary form of the disease. The growth of the tumour is accompanied by serous effusion from the choroid; the retina is detached and driven inwards; at first the vitreous makes room for the intrusion by parting with some of its fluid through the hyaloid into the aqueous chamber and so out of the eye; later, when the vitreous fluid is nearly all gone, further compensation is impossible; the lens, ciliary processes, and iris are driven forward, and the filtration angle is compressed just as in primary glaucoma. Glioma of the retina commonly leads in like manner to high tension, but the attack is of less violent character. Tumours of the iris may induce glaucoma by direct blocking of the filtration angle.

Intraocular hæmorrhage is sometimes the starting-point of a condition closely resembling primary glaucoma. It is important to distinguish these cases when possible, for hæmorrhagic glaucoma is less amenable to treatment.

Annular posterior synechia leads to secondary glaucoma by preventing the passage of the aqueous fluid forwards through the pupil. The iris is bulged forward by the accumulation of fluid behind it, the periphery of the chamber is abolished, and the eye is quickly lost by high tension unless an aperture be made in the iris by operation (see Fig. 4).

Perforating wounds and ulcers of the cornea lead to secondary glaucoma in certain cases through partial or total abolition of the anterior chamber, the iris being applied to the cornea. So long as the wound leaks, the eye remains soft; when leakage ceases, it becomes hard. The danger of this complication is increased when the injury is complicated by wounding of the lens.

Dislocation of the lens into the anterior chamber is sometimes followed by intense glaucoma. In such cases we can see that the iris is closely applied to the posterior surface of the lens and to the periphery of the cornea by the accumulation of fluid behind it. Lateral dislocation of the lens, as caused by a blow on the eye, is apt to lead to closure of the filtration angle by the pressure of the lens on the one side of the circle, and by the pressure of the displaced vitreous at the other.

Operations for the removal of cataract, whether by extraction or needling, are occasionally followed by secondary glaucoma. In all such cases careful examination shows compression of the filtration angle either by the swollen lens, or by the traction of membranes stretched across the pupil, incarcerated capsule, entangled iris or vitreous, or in some such way.

FIG. 4.—From an eye blinded by secondary glaucoma following neglected iritis. Exclusion of the pupil; accumulation of fluid in the posterior chamber; displacement of the iris; closure of the filtration angle.

Cyclitis in certain instances leads to secondary glaucoma, the ciliary body pouring out a morbid albuminous fluid which, unlike the normal aqueous, is unable to permeate the filtration angle and therefore tends to accumulate in the anterior chamber, which is deepened thereby. In this and in some other allied forms of the disease the high tension is due not to narrowing of the outlet, but to abnormal constitution of the fluid.

The rare form of glaucoma sometimes present at birth has been found to depend on congenital absence of the filtration angle, the condition being probably a fault of development rather than a product of disease. The glaucoma occasionally met with in eyes which present a partial or total absence of the iris has been found to

depend on blocking of the filtration area by a rudimentary structure, not visible in the living eye, which represents the iris.

It is obvious from the foregoing that the remote causes of glaucoma are very various, and include constitutional diseases; disorders of the respiratory, vascular, and nervous systems; injuries, morbid growths, congenital imperfections; and senile changes. The immediate cause of the high tension appears to be in all cases an obstruction which leads to accumulation of fluid within the eye.

SYMPTOMS.—The symptoms of glaucoma are numerous and complex, and vary considerably in different forms of the disease. They are the results of the pressure within the eye. In primary glaucoma we see these pressure changes in their uncomplicated form; in secondary glaucoma they are more or less modified by the presence of other changes in the eye. Before describing the clinical varieties of primary glaucoma it will be well to consider the various pressure symptoms from the point of view of their causation.

Increased tension of the eyeball is the leading symptom of glaucoma. Every medical man should know how to detect it:—The patient is told to look downwards; the surgeon, standing before him, steadies his hands by resting the outer fingers on the forehead, places the tips of his two forefingers on the upper eyelid, and feels the globe behind the margin of the cornea with gentle alternating pressure. He then feels the fellow-eye in like manner for comparison. It must be remembered that prominence of the eyeball is no evidence of increased tension, and that persons whose tension is normal frequently complain of a sense of fulness in the eyes. The following symbols are employed to describe variations of tension:—

TN: Tension normal.

T + 1 ?: Doubtful increase of tension.

T + 1: Slight but positive increase of tension.

T + 2: Considerable tension; the finger can slightly impress the coats.

T + 3: Extreme tension; the finger cannot dimple the eye by firm pressure.

T – 1 ?: Doubtful reduction of tension.

T – 1: Slight but positive reduction of tension.

T – 2, T – 3: Successive degrees of reduced tension.

Many attempts have been made with more or less success to replace the finger test by an instrument of precision—the tonometer. The instrument designed and used by the writer is so constructed as to make a known pressure on the surface of the sclera and to indicate the depth of the impression produced. For estimating doubtful changes of pressure it is a useful aid in many cases, but in daily work no instrument can replace the finger test.

Injection of the Eye.—A sudden access of high pressure, by embarrassing the internal circulation, and by reflex nerve action, causes intense engorgement of the external vessels, with more or less œdema of the conjunctiva, swelling of the lids, and sometimes even proptosis. In less violent attacks the visible injection is limited to the ciliary zone and the larger vessels which pass backwards from it. In glaucoma of gradual onset there is little or no visible injection of the eye beyond some enlargement of the main anterior ciliary vessels, arteries as well as veins. The ciliary arteries especially are more prominent and more difficult to empty by finger pressure than in health.

Pain.—Pain varies with the amount of vascular disturbance. In acute attacks it is sometimes extremely severe and radiates through all the branches of the fifth nerve. In slow non-congestive glaucoma it is often entirely absent, or occurs only in the last stage when congestion supervenes.

Cloudiness of the Cornea.—The lymph streams which traverse the normal cornea are hindered in their flow when the tension of the tunics is suddenly raised. A condition of slight œdema is set up, minute drops of fluid collecting beneath the anterior epithelium and between the more superficial fibres. It is this œdema of the cornea in its incipient stage that causes the appearance of rainbows round the light which many glaucoma patients notice at the beginning of an attack. When more pronounced it causes a visible cloudiness of the cornea and is in large part the cause of the extreme obscurity of vision which occurs during the height of an acute attack. The glaucomatous opacity of the cornea is distinguished from all forms of inflammatory opacity by the rapidity with which it appears and disappears in connection with changes of pressure in the eye. When high pressure is long continued the corneal epithelium may thicken and separate in vesicles or blebs; the opacity is then denser and more permanent. In glaucoma of very gradual onset the œdema of the cornea is entirely absent until the later stages of the disease are reached.

Anæsthesia of the Cornea.—During acute attacks, and in the later degenerative stages, the cornea partly loses its sensibility, probably through maceration and compression of the nerve filaments by the fluid collected beneath the epithelium, and their rupture when the epithelium is separated.

Dilatation of the Pupil.—In acute glaucoma the pupil is dilated. This depends probably on a lowering of the blood-supply to the iris produced by the rise of pressure and aggravated by the compression of the iris-base between the turgid ciliary processes and the cornea. The compression affects the nerves of the iris as well as the vessels, and the dilatation ultimately becomes permanent through paralysis and

atrophy of the sphincter. The oval dilatation seen in some cases probably shows that the compression has been more severe at some parts of the circle than at others. In chronic glaucoma, on the other hand, the vessels of the iris have time for a compensatory hypertrophy, and the blood-supply is maintained in spite of the gradually rising pressure in the eye; there is, moreover, little if any pinching of the iris base. In these cases, accordingly, the pupil dilates but little and sometimes not at all. It may remain of normal size and consensually active, even after the eye is blind. In those forms of secondary glaucoma which are associated with posterior synechia, there is of course no dilatation of the pupii.

Loss of Accommodation. — The range of accommodation is usually diminished by the onset of glaucoma, probably because the pressure in the eye makes the choroid more tense and increases its resistance to the contraction of the ciliary muscle. In the later stages the muscle atrophies.

Changes in the depth of the Anterior Chamber. —Shallowing of the anterior chamber is a common symptom in primary glaucoma, and in the allied forms which are induced by choroidal tumours and retinal hæmorrhage. Deepening of the chamber is characteristic of the secondary glaucoma of serous cyclitis and of congenital buphthalmos. In cases of the former type the lens is pushed forward by accumulation of fluid in the vitreous chamber; in the latter it is pushed backward by retention in the aqueous chamber.

Changes of Refraction.—Increase of pressure may influence the refraction in several ways. Supposing the eye to be naturally emmetropic, forward displacement of the lens induces myopia, backward displacement hypermetropia. The elongation of the globe which occurs in the glaucoma of children induces myopia of high degree. In the majority of cases the refraction, if altered at all, is increased. It is often found higher during a glaucomatous attack than before or after. Moderate pressure long continued appears in some cases to induce astigmatism of exceptional kind, the horizontal meridian of the cornea becoming more sharply curved than the vertical. The opposite condition, commonly found after an upward iridectomy, is of course due to a flattening of the vertical meridian induced by the marginal incision.

Excavation of the Optic Disc.—Under continued pressure the optic papilla is transformed into a cup. The lamina cribrosa,—the sieve-like part of the sclera which gives passage to the optic nerve fibres,—being the weakest spot in the wall of the eye, is displaced backward, together with the nerve-fibres and blood-vessels which it supports. The firmer ring of sclera around it limits the area of excavation. The nerve-fibres, being bent and stretched over the unyielding margin, suffer atrophy and loss of bulk, and a deep undermined cup results. Cupping is not found during or after a first attack of acute glaucoma, for the atrophy takes time. On examining a cupped disc with the ophthalmoscope by the indirect method, and making small lateral movements of the object lens, we see that the vessels in the plane of the retina have a greater apparent movement than those at the bottom of the cup, and seem to outrun and travel in front of them. By the direct method we see the difference of refraction between the margin and the bottom of the cup, and the depth of the latter may be roughly estimated by remembering that a difference of three dioptres corresponds to a difference of level of about 1 mm. The floor of the cup is paler than the normal disc and more distinctly cribriform, through atrophy of the nerve-fibres. The sides are more or less hidden by the over-hanging margin, so that the vessels seen on the floor are lost to view as they ascend the sides, and reappear changed in number and position as they bend round the margin to gain the retina. Around the margin there is usually some atrophy of the choroid visible as a zone of lighter colour than the adjacent fundus. The vessels are more or less displaced toward the nasal side of the cup. When associated with posterior staphyloma, as in high myopia, the glaucoma cup is larger in diameter than usual; its sides are little if at all undermined; its vessels are attenuated by elongation and comparatively free from sharp bends or curves. The glaucoma cup is to be distinguished from the so-called physiological cup—a central depression often present in the healthy disc—by the fact that it involves the whole area of the disc. From the shallow excavation of simple atrophy it is to be distinguished by its depth and by the sudden bending and interruption of the vessels at its margin. These distinctions, however, are not always easy to make, and when simple atrophy occurs in a disc in which there is already a large physiological cup, the resulting condition may closely resemble the typical glaucoma cup.

Pulsation of Retinal Vessels.—The pressure on the retina obstructs the entrance of the arterial and the exit of the venous streams. Hence the arteries are apt to be incompletely filled, the veins congested. Arterial pulsation, rarely visible in a healthy eye, is often to be seen in the area of the disc, or may be induced by light finger pressure on the globe. The veins are rhythmically compressed during each arterial pulsation by the pressure transmitted to them through the vitreous. This venous pulsation occurs only in a small portion of the vein close to its point of exit, for the blood-pressure is lowest here, and compression at this point prevents the expulsion of the blood from the adjacent part of the vessel. It is

often visible in eyes of normal tension, but not so often as in glaucoma. Capillary hæmorrhage may result from the obstruction of the retinal circulation. Aneurysmal dilatations of the arteries and beadlike varicosities of the veins are occasionally seen.

Impairment of Vision.—Vision is affected in several ways by the pressure in the eye.

The transient œdema of the cornea which occurs when glaucoma is threatening causes temporary obscuration of vision in the daytime, and an appearance of a ring of rainbow colours around every luminous flame at night. The characters of this rainbow vision are these: The flame is seen with nearly normal clearness; around it is a dark non-luminous zone, the breadth of which on each side of the flame corresponds to an angle of 4° to 5°. Surrounding this is the coloured zone, which has an angular breadth of 2° to 2°·5, and a total diameter of about 10° to 11°. In the coloured zone the whole of the colours of the spectrum are visible, the violet being invariably at the inner, the red at the outer border. The zone is not altered either as to size or as to position of the colours by the use of convex or concave lenses, and it is not altered by variations in the diameter of the pupil. It is perceived not only in direct vision, but also, though with less distinctness, when the image of the flame falls on other parts of the retina than the yellow spot. An appearance corresponding in all respects with the rainbow vision of glaucoma can be induced experimentally by the application of a single drop of a 0·125 P.C. solution of hydrochlorate of erythrophleine to the eye; at the same time a faint haze of the corneal epithelium occurs. This experiment, while it clearly shows the dependence of rainbow vision on a disturbance of the corneal epithelium, proves also that it is not necessarily dependent on an excess of pressure in the eye. The much-dreaded rainbows, therefore, are not invariably a sign that glaucoma is approaching. They occur also in some congestive states in which no increase of tension is discoverable.

Further, the sensibility of the retina is lowered when an excess of pressure falls upon it, as any one may discover for himself by making finger pressure on the eyeball—an experiment not to be made frequently or incautiously. Sensibility may be abolished by pressure, over the whole area of the retina, but less easily in the region of the macula than elsewhere. Acute glaucoma is attended with a more or less complete retinal paralysis of this character. It appears to depend on a disturbance of the circulation in the retinal vessels, and perhaps in the choroidal plexus, on which the rods and cones of the retina depend for their nutrition. Chronic glaucoma is attended by retinal paralysis of a different kind. The field of vision fails at the periphery, while at the centre it remains for a while intact. This peripheral contraction of the field is discoverable at first only in a subdued light. It commonly begins at the inner or nasal margin of the field, involves the inner, upper, and lower portions before the outer, and slowly advances till it has reduced the sentient area to a small oval or slit-like form extending outwards from the fixation point to the blind spot and beyond it. It next involves the fixation point and the adjacent area, so as to leave only a small eccentric area of vision extending outward from the blind spot, corresponding to a small area of the retina extending inwards from the optic disc. Ultimately this fails also and the eye is blind. This contraction of the field is probably the expression of progressive damage of nerve-fibres in the excavated disc, those belonging to the temporal half of the retina suffering earlier than those belonging to the nasal half; those passing to the periphery earlier than those supplying the macular region. The contraction does not always follow the typical course here described, but the comparative immunity of the central and outer parts of the field during the failure of the remainder is found in a very large majority of cases. This characteristic, together with the relatively sharp line of demarcation between the sentient and non-sentient areas, and the relatively good retention of colour vision in the former, help to distinguish the contracted field of chronic glaucoma from that of primary atrophy of the optic nerve. When the field is tested by means of very minute test objects defects are found which would otherwise escape detection, and these defective areas, wherever situated, appear to be in direct continuity with the blind spot of the optic disc. The two types of retinal paralysis are often combined. An acute glaucoma initiates the loss of vision by obstructing the circulation; prolonged pressure completes it by excavating the disc. Timely removal of the pressure will do much to restore what is lost through recent interference with the blood-supply, but the loss due to atrophy of nerve-fibres is permanent.

Clinical Varieties of Primary Glaucoma.—*Acute primary glaucoma* is apt to be mistaken for an inflammation of the eye, an attack of erysipelas, or a neuralgia. In some cases it arises without known cause and without warning of any kind. More frequently it is preceded by one or more transient attacks of dimness and rainbow vision, and is attributable to chill, exhaustion, loss of sleep, or some other of the exciting causes which have already been discussed. In some cases it shortly follows the use of atropine or some other dilator of the pupil. Pain begins in the eye more or less suddenly, increases from hour to hour, and extends over the side of the head and face. In the severest cases vomiting occurs and the patient is much prostrated. Vision is much

impaired. When summoned to the case the surgeon probably finds the conjunctiva much injected, and the eyelids, in severe cases, somewhat swollen. The subconjunctival vessels are engorged, especially as regards the circumcorneal zone and the main trunks connected with it. The cornea is more or less wanting in polish, and if high tension has already persisted some days its sensitiveness to touch is diminished. The pupil is more or less dilated, totally inactive, and sometimes somewhat oval; the dilatation in conjunction with the injection of the eye should at once arouse a suspicion of glaucoma. The anterior chamber is usually shallow. The eyeball is decidedly too hard under the finger test, and this fact establishes the diagnosis. The vision of the eye may in the course of a few days, or even sooner, be reduced to a mere perception of light near to the middle of the field, or it may be totally extinguished. Ophthalmoscopic examination is usually prevented by the cloudiness of the cornea; but, if visible, the retinal arteries will be seen to be somewhat reduced in size and perhaps pulsating, the veins engorged, and, at their point of emergence from the disc, collapsing with each arterial pulse. Hæmorrhages are sometimes discoverable in the retina. Such an acute attack, unless relieved by treatment, will commonly last for several weeks and then slowly subside, but though the more prominent symptoms disappear, the eye remains hard and the remnant of sight is gradually lost. The most acute cases have been termed fulminating from the violence of their onset; the less acute form a connecting link with the subacute variety. The more acute the attack the greater the need for prompt and efficient treatment if sight is to be saved.

Subacute primary glaucoma resembles the acute form, but is milder and for a while intermittent. At first there may be merely temporary periods of mistiness and rainbow vision. Sooner or later the recurrences become more frequent, the intermissions less complete, and a persistent congestive glaucomatous condition is established. Blindness comes on less rapidly than during acute glaucoma, but not less surely. These congestive forms of the disease are spoken of by many writers as inflammatory glaucoma, and it is true that inflammation attends their course; but, seeing that the condition is essentially one of mechanical obstruction, and not of primary inflammation, the term is apt to mislead, and is on that account objectionable.

Chronic primary glaucoma (simple glaucoma) is apt to pass unnoticed even by the patient in its first stage, and may be mistaken by the medical man for simple atrophy of the optic nerve. It begins almost imperceptibly, progresses slowly with little tendency to exacerbation or remission, and leads, in the course of months or years, unless arrested by treatment, to total blindness. The patient is usually at least fifty years of age. He complains of failing sight in one eye or in both. One eye is usually affected first, the other almost always follows sooner or later. There is usually no history of periodic obscuration, and the onset of the disorder cannot be exactly dated. It sometimes appears to have been preceded by a period of mental strain of some kind. Externally, the eyes exhibit little or nothing amiss. The chief anterior ciliary vessels may be somewhat enlarged, the anterior chamber rather shallow. One or both pupils may be somewhat dilated, but this is not a constant symptom. An eye already blind through chronic glaucoma may have a pupil which is equal to that of the seeing eye, and which acts consensually with it. The lens, on simple inspection, may appear to be more or less cataractous, and yet, when examined with the ophthalmoscope, prove to be quite transparent. Mistakes of diagnosis from this cause are not infrequent and are very unfortunate, for a delay which may be wise in the case of cataract is disastrous in the presence of glaucoma. The diagnosis depends chiefly on the tension of the eye, the field of vision, and the condition of the disc. The tension is increased, but in some cases, especially in the earlier stage, the increase may be very slight and at times absent. When possible, it should be tested at different times of day. The same eye may be quite normal to the touch at one time, distinctly tense at another. It is usually towards night, when the patient is fatigued, that the increase is most perceptible. In the later stages the eye may become extremely hard, and yet, by reason of the slow progress of the disorder, there may be no pain and little external sign of disease. The field of vision will show, when mapped by means of the perimeter, the characteristic and gradually increasing contraction which has been described. The optic disc will show the typical glaucoma cup. The course of the disease is slow, and in exceptional cases may even extend over many years before the stage of complete blindness is reached.

Absolute Glaucoma.—When the eye has lost perception of light in all parts of the field the glaucoma is said to be absolute, and in this stage the ultimate effects of high pressure are gradually developed. If the glaucoma have been acute or subacute, the disturbances of the eye remain very obvious. The external vessels are large and tortuous, and stand out prominently in the atrophied conjunctiva; the cornea is hazy, its epithelium thickened, and its sensibility lowered or abolished; the iris, if visible, is seen as a narrow circle by reason of the wide dilatation of the pupil, and it may be visibly in contact with the cornea; enlarged blood-vessels may be visible on its surface; the lens degenerates and

ultimately becomes opaque, sometimes conspicuously white or yellow. Pain is apt to continue or to recur from time to time, and it is for this that the patient seeks relief. Subjective visual sensations may delude the patient into hoping for some recovery of sight. If the glaucoma have been chronic throughout, without pain or congestion, the appearance of the eye may be nearly normal even in the absolute stage, but the high tension, and the excavation of the disc, at once reveal the cause of the blindness. Sooner or later, however, such an eye is apt to suffer attacks of pain and congestion which demand relief, and this relief can be given more speedily and certainly by removal of the eye than in any other way. The last stage of glaucoma is marked by gradual thinning and extension of the wall of the eye, commonly in the region of the equator, sometimes in the ciliary zone. Rupture of the globe occasionally completes the destructive process.

Secondary Glaucoma.—The varieties of secondary glaucoma need not be further described The diseases of the eye which are liable to this complication have been enumerated. Increase of tension is the essential sign of its onset. Vascular injection, pain, and impairment of vision, if already present, are aggravated by the access of high pressure in the eye. The pupil, if it be free, dilates. The field of vision, if not already lost, contracts. The optic disc in course of time suffers excavation, though this latter change is commonly hidden from observation in the living eye, and discoverable only after the eye has been removed.

Treatment. — The successful treatment of glaucoma depends essentially upon lowering the increased pressure within the eye, and the measures by which this can be effected must be employed in the earlier stages of the disease if permanent loss of sight is to be avoided. When the eye is blind, treatment can avail nothing beyond the relief of pain or the removal of disfigurement, and in most cases these ends are attainable more certainly, speedily, and safely by complete removal of the useless organ than in any other way.

Glaucoma usually calls for operative treatment, for it is seldom possible, otherwise than by operation, to re-establish a permanent and sufficient outlet for the intra-ocular fluid. There are many cases, however, in which certain auxiliary lines of treatment are of great value, and there are a few in which benefit of long duration may be obtained by these means alone. It is chiefly primary glaucoma which we have here to consider.

Myotics.—Drugs which contract the pupil, of which those chiefly used are physostigmine (eserine) and pilocarpine, sometimes rapidly relieve the high tension. They are the antagonists of atropine, which dilates the pupil and, where predisposing conditions exist, induces or aggravates high tension. It is to be noted that neither myotics nor mydriatics cause any decided change of tension in normal eyes. Their action in relation to glaucoma appears to depend mainly on the abnormal position of the iris. Eserine and pilocarpine, by contracting the sphincter of the pupil, attenuate the iris, flatten out its folds, and pull upon its peripheral insertion. These changes tend to reopen the filtration angle when it is narrowed or compressed. Accordingly, it is chiefly in the comparatively mild attacks which come and go in the early stage of the disease that we can rely upon their action. In severe acute attacks they may be very useful if used without delay, but in these cases the iris base is firmly compressed, it forms adhesions early, and the sphincter of the pupil soon loses its contractility; hence the stage in which myotics can relieve these violent attacks is soon at an end. In chronic non-congestive glaucoma some lowering of tension may be obtainable over a considerable period of time by a continued use of eserine or pilocarpine, but the improvement is seldom great or permanent. Of these two drugs, if used in equal quantity, eserine is much the more active and the more irritating; a one-half per cent solution of sulphate of eserine in distilled water (roughly two grains to one ounce) is amply strong enough for any purpose, and in most cases a much weaker solution is better. A one or two per cent solution of nitrate or hydrochlorate of pilocarpine appears in many cases to be equally effective and to be better tolerated. These drugs should be used in the minimum amount, and with the minimum frequency which suffice to contract the pupil and to keep it contracted: usually one drop once or twice a day, sometimes more frequently, and in some cases of chronic glaucoma, at longer intervals. In cases where slight temporary attacks occur at considerable intervals of time, the drops should be used only as often as is necessary to banish them. In cases in which the contractility of the pupil is entirely lost myotics are probably useless, and in these same cases atropine is probably harmless, and sometimes perhaps beneficial. When atropine cannot dilate the pupil it never probably raises the tension, and in those forms of secondary glaucoma characterised by serous exudation into the aqueous chamber, with deepening of the anterior chamber, atropine tends to lessen the inflammation and thereby to restore the normal tension. Apart from their influence on the iris and ciliary muscle, however, the action of these drugs is not yet completely known, and in some cases of glaucoma, especially during treatment after operation, and in some forms of secondary glaucoma, the choice between a myotic and a mydriatic is not easy to make; the one or the other must be used tentatively, and its action carefully watched.

Cocaine, like every other dilator of the pupil,

may, under predisposing conditions, induce or aggravate glaucoma. It has, however, the power of contracting the ciliary blood-vessels and diminishing the sensibility of the ciliary nerves—effects which tend to lower the intra-ocular pressure. It is often useful, therefore, to combine cocaine with eserine or pilocarpine in such proportions that, while the cocaine helps to subdue the vascular congestion, the myotic keeps the pupil contracted.

Morphine given subcutaneously, or by the mouth, will sometimes rapidly cut short an acute attack, and is useful in many cases. It eases pain, promotes sleep and contraction of the pupil, lowers the blood-pressure, and probably diminishes for a time the secretion from the ciliary processes. *Antipyrin* and some other drugs of the same class are useful in the same way; they can be repeated with more freedom than morphine, but have a less positive influence over the tension of the eye.

Sleep, even though of very short duration, and occurring without the aid of any drug, often dispels the mild premonitory attacks by which primary glaucoma is ushered in. During sleep the pressure in the cerebral vessels falls and the pupil contracts. *Warmth*, *food*, and *rest* relieve, just as cold, hunger, and fatigue induce these early and slight attacks.

Aperients are often needed, and sometimes have a well-marked effect in lessening the fulness of the cerebral vessels and the pressure in the eye. The habit of straining at stool should be expressly forbidden; it congests the head and tends to aggravate the glaucomatous condition.

Ice applied over the closed lids, in the form of ice compresses or in a thin rubber balloon, is sometimes useful in conjunction with other measures in presence of acute congestion.

By a judicious combination of these palliative measures, especially by contracting the pupil, relieving pain, and inducing sleep, we can frequently, especially in early acute cases, obtain great improvement for a time, and thereby postpone the time at which an operation is imperative; we can then operate under more favourable conditions; but the temporary benefit obtained in this way is likely to do more harm than good if it leads to undue delay of operative treatment.

Operative Treatment.—The object with which we operate is to obtain a permanent and sufficient outlet for the intra-ocular fluid. The most trustworthy operation in the large majority of cases is *iridectomy.* The improvement of vision obtainable by iridectomy varies with the type of the disease: it is greatest in the acute form, smallest in the chronic. An acute attack may in the course of a few days reduce vision from its normal condition to a bare perception of light; in such a case an iridectomy promptly performed may restore it nearly, if not quite, to its former condition. A chronic glaucoma, on the other hand, may advance slowly and insidiously for a year or more, excavating the disc and contracting the visual fluid, but causing little change in the acuteness of central vision as indicated by test types. An iridectomy in such a case may enlarge the visual field a little; it may arrest the progress of the malady; it may preserve the vision which still remains but cannot greatly improve it. The urgency for operation varies, of course, with the acuteness of the disease. In acute glaucoma, unless improvement is rapidly obtained by the measures already discussed, iridectomy should be performed without delay. The prostration of the patient, though severe, is no reason for delay, for an iridectomy is the surest means of giving ease and sleep as well as of saving the eyesight. If light-perception be already completely lost we cannot hope for restoration of good vision, but if it has not been lost many days, it is still right to operate at once, for there is still the chance of recovering some amount of vision. In subacute glaucoma also it is important to operate early, for each recurrence of the acute symptoms is likely to render a perfect operation more difficult of attainment, and a perfect recovery of vision less probable. In such cases it is best to cut short the acuter symptoms if possible by palliative treatment and to operate during a quiescent period. In chronic non-congestive glaucoma the benefit obtainable by iridectomy is less obvious and less certain; positive improvement of vision is seldom to be hoped for; the object of the operation is to prevent further loss. Even this is not always achieved. The operation sometimes fails to arrest the course of the disease, and occasionally accelerates it. When repeated examinations of the eye at intervals of a week or two, or longer, have shown that the visual field continues to contract, and that the tension remains in excess, in spite of a careful trial of myotics, then it is usually our duty to advise operation. It is the only means by which the eye can be saved from certain blindness, but is not a certain means, and this should be carefully explained to the patient, or in some cases to his friends, before it is undertaken. If both eyes are affected, but both still retain some sight, the worse should be operated on first. The result may decide the treatment of the other. Some surgeons advise their patients, especially if there be already great contraction of the field, to accept the gradual approach of blindness rather than the risk of operation, and in some cases, where the probable duration of life is short, this is certainly the right course, but as a general rule our duty is to advise operation so long as the eye retains sight worth saving. Cases of definite arrest of chronic glaucoma, with retention of useful vision over many years, are known to all experienced operators. The benefit ob-

tained must be estimated afterwards rather by the field of vision which is retained than by the central acuteness—that is, rather by the perimeter than by the test types.

Mode of Performing Iridectomy.—The operation consists in making an incision which opens the anterior chamber near to its periphery, and removing as completely as possible the corresponding portion of the iris. When the eye is painful a general anæsthetic is usually required; in chronic non-congestive glaucoma cocaine will usually suffice, and we have then the advantage that we can control the position of the eye by letting the patient look at a lighted candle held in the requisite position. The incision is made with a small linear cataract knife. It is usually placed at the upper part of the cornea. The puncture is in the sclera at least 1 millimetre from the corneal margin; the counter-puncture is at a corresponding point; the distance between puncture and counter-puncture externally in a straight line is from 7 to 9 millimetres, according to the size of the cornea and the depth of the anterior chamber. When the iris lies very close to the cornea it is impossible to make a full-sized incision. In passing across the chamber the knife is kept parallel with the plane of the iris; in cutting out the edge is turned somewhat forward, but must come out well in the sclera and raise a conjunctival flat. The iris is seized with the small forceps, drawn out through the incision, and lightly pulled to the right and to the left, so that its base may be disengaged from the cornea. It is then divided with scissors close to one end of the incision, drawn towards the other end, and if possible torn at its root; is again drawn away from the angle of the wound so that it may not be pinched and incarcerated; and is removed with a second snip of the scissors. Some operators make the incision with a broad lance knife instead of the cataract knife. In the writer's opinion, the cataract knife enables the surgeon to make a more peripheral incision than can be safely made with the lance, and to modify its length and position, according to the space available, after the point has appeared in the chamber, by making the counter-puncture a little farther forward or backward as may be found practicable. Some operators place the incision at the part of the circle where the iris appears to respond most readily to myotics, on the ground that removal of the iris-base is more likely to be attained here than elsewhere. For a laterally placed incision the lance knife must be used. The upward incision has the advantage that it can be made with the linear knife, and that the coloboma lies under the upper lid. In the writer's opinion, it is advisable in most cases to slacken the eye by a scleral puncture immediately before making the iridectomy. (See later.)

A successful iridectomy permanently removes the excess of tension either by causing the normal filtration outlet to reopen or by establishing an abnormal one. In a recent acute glaucoma a good iridectomy permits an escape of blood from the turgid ciliary vessels, and an escape of clear fluid from aqueous and vitreous chambers. The ciliary processes recede and cease to compress the base of the iris, and with the re-establishment of the anterior chamber the filtration angle reopens: the normal outlet resumes its function. This has been proved by the examination of eyes cured years previously by iridectomy. The absence of the iris segment appears to act as a safeguard against the recurrence of a similar blockade. In congestive glaucoma of longer standing restitution of the filtration angle is often unattainable, but even in such cases a well-made iridectomy will sometimes free the ligamentum pectinatum in the region of the wound by tearing away the iris from its root. In many of these cases, however, and in chronic glaucoma also, the success of an iridectomy depends on the formation of an artificial filtration channel: a minute, permanent, corneo-scleral fistula. The lips of the wound, especially the inner lips, do not unite completely, but remain more or less separated by the prolapse between them of a fold or tag of iris. The aqueous continues to escape at this point into the subconjunctival tissue, and is thence absorbed. The overlying conjunctiva presents an œdematous or pearly appearance, and is more or less elevated by the collection of fluid beneath it. Finger pressure carefully applied to the eye day after day during the healing process aids in keeping the tension low, and probably promotes the formation of such fistulæ. Even weeks after an iridectomy firm pressure with the finger will sometimes cause an immediate visible extrusion of fluid beneath the conjunctiva, with slackening of the globe. In chronic glaucoma, and in the later stages of the congestive forms, a slightly fistulous scar affords the best, if not the only, guarantee against a speedy return of the glaucoma, and is therefore a result to be desired. After an iridectomy for glaucoma there is usually a considerable flattening of the cornea in the meridian, which is at right angles with the cicatrix; in other words, there is a certain degree of ectasia in the region of the cicatrix which probably increases the distance between the ciliary body and the lens margin, and thereby lessens the danger of complete compression of the filtration angle in the future.

An iridectomy may fail in several ways. Profuse hæmorrhage within the eye at the time of the operation, or during the following few days, is perhaps the one over which we have least control. It is chiefly to be feared where the glaucoma is known to have been preceded by intra-ocular hæmorrhage; it may happen in spite of the greatest care where there

is advanced degeneration of the blood-vessels; it may be induced by violent coughing, sneezing, or straining of any kind; it may depend on direct injury of the ciliary processes by a deeply placed incision. Its avoidance depends largely on careful regulation of the patient's condition with regard to the action of bowel and kidneys, sleep, and absence of cough; on gentleness in operating, and on rest and tranquillity during the following few days. The worst form of hæmorrhage is the retro-choroidal; it causes extrusion of the lens and vitreous through the incision, and calls for prompt removal of the eye. It is very rare. Failures of any kind through hæmorrhage are quite exceptional. More frequently an iridectomy fails by effecting neither a reopening of the filtration angle nor the formation of a vicarious channel. Thus an incision which lies entirely in the cornea, or nearly so, closes too quickly and firmly, giving an inextensible cicatrix which affords no drainage. Again, a very shallow chamber with high tension involves danger to the lens. As the aqueous escapes the lens is driven forward by pressure from behind, and may be ruptured against the back of the knife through the intervening iris; or without being actually injured, it may be driven forwards so forcibly after the operation as to block the wound and prevent the drainage of the eye; it may even be extruded through the wound during the following twenty-four hours. When, after an apparently perfect operation, we find on the following day that the iris is in contact or nearly so with the cornea, the wound not leaking, and the eye hard, we have the formidable condition known as malignant glaucoma. The displaced lens is blocking the wound, and the best chance of saving the eye lies in making a scleral puncture as described below, and following it at once by steady pressure on the centre of the cornea by means of a curette or other smooth instrument, continued until the anterior chamber is re-established by the entrance of fluid between iris and cornea. For this a general anæsthetic is likely to be required. If this manœuvre fail, or if the lens be injured as well as displaced, extraction of the lens affords a last chance of saving the eye, the posterior capsule being divided at the same time, so as to establish free communication with the vitreous chamber. A shallow chamber after iridectomy need excite no apprehension so long as the tension of the eye remains low.

Sclerotomy.—This operation opens the anterior chamber by an incision more or less resembling the first act of an iridectomy, and leaves the iris intact. Experience has shown that every form of primary glaucoma will yield in some cases to sclerotomy, but it has shown also that sclerotomy is on the whole less trustworthy than the older operation, for the reason that the iris is apt to occlude the wound during the healing process, and that in any case the filtration angle remains more liable to occlusion than after the removal of the iris-segment. The chief use of sclerotomy is as a supplement to iridectomy when the latter fails to permanently reduce the tension. In the absence of the iris-segment it is a simple operation, and is generally preferable to a second iridectomy at the opposite side of the circle. Sclerotomy, like iridectomy, may sometimes be advantageously preceded by a scleral puncture. For glaucoma with deep anterior chamber, as in serous cyclitis and congenital buphthalmos, *paracentesis of the cornea* is usually preferable to iridectomy. In such cases the incision must be of small extent, and repeated if necessary several times, each time at a different part of the cornea. For secondary glaucoma after cataract extraction, sclerotomy combined with division of any membrane which is stretched across the pupil is usually the best remedy.

Scleral Puncture or Posterior Sclerotomy.—The eye is turned inwards so as to expose the outer part of the sclera. The surgeon, taking a Graefe knife in one hand and forceps in the other, seizes the conjunctiva a little below the horizontal meridian, and slides it downwards a little over the sclera. Keeping the back of the knife towards the cornea, and the point directed towards the centre of the globe, he punctures the sclera at a point about six millimetres from the corneal margin and a little below the horizontal meridian. After entering about ten millimetres the knife is rotated on its axis, so as to give a gaping wound through which fluid escapes, and slowly withdrawn. The conjunctiva is then allowed to slide back into its place so that the conjunctival and scleral wounds are not directly continuous. This operation is useful chiefly as a preliminary or adjunct to iridectomy or sclerotomy. Used alone, although it gives immediate relief of tension and corresponding improvement of vision, it rarely if ever effects a permanent cure. A scleral puncture made immediately before a glaucoma iridectomy, especially where the chamber is shallow and the eye very hard, facilitates the making of a good incision, and thereby diminishes the risks and the causes of possible failure which attend an iridectomy under these circumstances. It occasionally, however, reduces the tension of the eye to such an extent as to hinder rather than help the performance of the iridectomy. In such cases, which in the writer's experience are extremely rare, the scleral puncture will suffice for temporary relief, and the iridectomy must be postponed until a more normal tension is restored. In very advanced and doubtful cases, where the possibility of recovering useful sight is uncertain, it is well to make a scleral puncture as a test operation in the first instance, and if the result be encouraging, to make an iridectomy a few days later.

Other Substitutes for Iridectomy.—In cases of

annular posterior synechia with bulging iris, it is sometimes impossible to pass the linear knife between the iris and the cornea. It may then be passed through and behind the iris instead of in front of it, and will, in cutting out, re-establish the desired communication between posterior and anterior chambers. Quite a small aperture in the iris may suffice in such a case to permanently relieve the tension.

Incisions and punctures which divide the ciliary region in a more or less meridial direction, and which, if they involve the corneo-scleral junction, will open the aqueous and vitreous chambers simultaneously, were formerly and are still occasionally practised. Such incisions are capable of giving immediate relief and of depleting the ciliary body, but they are not comparable in safety or certainty of result with a well-made iridectomy.

Stretching and rupture of the infra-trochlear nerve, and excision of the superior cervical ganglion of the sympathetic, are methods occasionally employed where other treatment has failed. The evidence in their favour is not very strong.

After-Treatment.—The essential points in the after-treatment are to obtain quietude and sleep, to apply the necessary dressings with little pressure, and to guard against stooping and all sudden or straining movements. So long as the chamber is empty, or the wound obviously leaking, the patient should remain in bed. Finally, the refractive condition of the eye must be tested and corrected with special regard to the astigmatism which the operation commonly sets up, and the patient must be carefully instructed to avoid such conditions or habits of life as might lead to a recurrence of his malady.

Glaucosis.—Blindness produced by glaucoma (*q.v.*).

Glaucosuria.—A morbid condition of the urine in which it has a green colour.

Gleet.—Chronic gonorrhœa, chronic urethritis, or chronic blennorrhœa. *See* URETHRA, DISEASES OF (*Gonorrhœa*); GONORRHŒAL INFECTION.

Gleichenburg. *See* BALNEOLOGY (*Austria, Alkaline*).

Glenard's Disease. *See* ENTEROPTOSIS.

Glengore. *See* SYPHILIS (*Historical*).

Gleno-.—In compound words *gleno-* means relating to a shallow joint or articular cavity (*e.g.* gleno-humeral) or to the socket of the eye.

Gliadin.—Vegetable gelatin, the tenacious mass, insoluble in water, left as a residuum after the evaporation of a strong alcoholic solution of gluten.

Glioma.—A colloid neoplasm of the nervous system or a sarcomatous tumour of the retina. *See* BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Hæmorrhage, Pathology*); GLAUCOMA (*Causes of Secondary Glaucoma*).

Gliomatous Arthropathy.—Syringomyelia, a disease in which there are new formations in the spinal cord consisting of glial cells, and in which joint affections are common. *See* JOINTS, DISEASES OF (*Associated with Lesions of the Nervous System, Syringomyelia, Pathological Anatomy*); SYRINGOMYELIA.

Gliosis.—A morbid condition in which embryonal neuroglial tissue forms and then breaks down into cavities (on account of hæmorrhage, etc.); it may occur in the cerebrum (*see* INSANITY, PATHOLOGY, HYPERTROPHIC NODULAR GLIOSIS) or in the spinal cord (*see* SYRINGOMYELIA).

Globin.—One of the derivatives of hæmoglobin. *See* PHYSIOLOGY, BLOOD (*Decomposition of Hæmoglobin*).

Globule.—A small spherical mass, such as a blood or colostrum corpuscle or the body extruded from the ovum during maturation (*polar globule*), or a pill or capsule containing a medicinal substance.

Globulin.—A simple, native proteid; it is insoluble in water and so differs from albumin; *globulinuria* signifies the presence of globulins in the urine. *See* PHYSIOLOGY, PROTOPLASM (*Classification of the Proteids*); URINE, PATHOLOGICAL CHANGES (*Albuminuria*).

Globulose.—A proteose or proteid with a less complex molecule than globulin. *See* PHYSIOLOGY, PROTOPLASM (*Proteids*).

Globus Hystericus.—The feeling, as of a ball rising in the throat, often complained of in the onset of a hysterical fit. *See* HYSTERIA.

Glomerulus. *See* PHYSIOLOGY, EXCRETION (*Kidney, Structure*); KIDNEY, PHYSIOLOGY OF.

Glonoin Oil.—Nitroglycerin or Nobel's Blasting Oil; the name has originated in the chemical formula of the substance—Gl representing glyceryl, C_3H_5—which is $GlONO_3$. *See* NITROGLYCERIN.

Glossalgia.—Pain in the tongue; glossodynia.

Gloss Anthrax.—Anthrax affecting specially the mucous membranes of the mouth. *See* ANTHRAX (*in Animals*).

Glossitis.—Inflammation of the tongue. *See* Tongue (*Inflammatory Affections*).

Glossocele.—Protrusion of the tongue, from disease or on account of congenital hypertrophy (sometimes unilateral).

Glossodynia.—Pain in the tongue; glossalgia.

Glosso-labio-laryngeal Paralysis.—Bulbar paralysis. *See* Paralysis (*Paralysis with Atrophy of Muscles, Chronic Nuclear Bulbar Paralysis*).

Glosso-Pharyngeal Nerve. *See also* Brain, Physiology of (*Ninth Cranial Nerve*); Physiology, Nervous System (*Cranial Nerves, Ninth Pair*); Tabes Dorsalis (*Symptomatology, Affection of Cranial Nerves*).

This nerve comes out by five or six filaments from the upper part of the medulla, in the groove between the olivary and restiform bodies, and the fibres can be traced to three nuclei, the glosso-pharyngeal nucleus proper, the funiculus solitarius, and some of the fibres, probably motor, pass into the upper part of the nucleus ambiguus, which mainly belongs to the vagus. (Some authorities consider it difficult accurately to differentiate the nuclei of origin of this nerve from those of the vagus, and further hold that this nerve ought to be described as a part of the vagus.) The nerve passes out through the middle division of the jugular foramen, along with the spinal accessory and vagus nerves, but separated from them by a special sheath of fibrous tissue. In the foramen it is external, and also in front of the other nerves, and has on it two ganglia, the jugular and the petrosal, which resemble the spinal ganglia as regards their constituent cells.

Following Quain's description, the nerve appears below the internal carotid and jugular vein, and is directed downwards over the carotid artery, and beneath the styloid process and the muscles connected with it, to the hinder border of the stylo-pharyngeus muscle; then curving forwards, it crosses over the outer surface of this muscle, and passing beneath the hyoglossus ends in branches for the posterior part of the tongue. The jugular ganglion is situated in the jugular foramen, and includes only the lower fibres of the nerve. The petrosal ganglion is situated in a small depression in the petrous part of the temporal bone, and from it the nerve of Jacobson and the branches to the vagus and sympathetic arise.

The nerve supplies *sensory branches* to the back of the tongue, the pharynx, Eustachian tube and middle ear. Taste fibres for the posterior third of the tongue and neighbouring gustatory mucous membrane are carried by this nerve. The fibres probably in most cases leave the glosso-pharyngeal by way of Jacobson's nerve, the small superficial petrosal, and the otic ganglion, to reach the third division of the trigeminus.

The nerve supplies *motor fibres* to the stylo-pharyngeus muscle, and possibly middle constricture of the pharynx, very probably along with the vagus. The nerve also supplies *secretory and vaso-dilator* fibres to the parotid gland through the otic ganglion and the auriculo-temporal nerve, and it is connected with the vagus, the third division of the fifth, the seventh, and the sympathetic.

Etiology and Clinical Features.—The nuclei of the nerve might be injured in the medulla by hæmorrhages, acute softening, sclerosis, or tumours, and at the point of exit of its constituent filaments, by syphilitic disease, aneurysm, or meningitis, and in the peripheral course of the nerve by thrombosis of the jugular vein, or inflammatory swellings, tumours, and so forth compressing the nerve.

The nuclei suffer, but never as an isolated lesion, and in acute and chronic bulbar paralyses other nuclei are always included, and it is hardly conceivable that in a lesion affecting the nerve, whether intra- or extra-cranially, that the vagus at least could escape.

The *clinical features* can hardly be stated with any degree of certainty, as isolated lesions of this nerve are not obtainable in sufficient numbers to supply data. If the nerve is injured beyond the ganglia then taste should be lost in the posterior third of the tongue. If the ordinary sensory and motor fibres are injured the pharynx will be insensitive, and food is swallowed with difficulty, partly due to this loss of sensation, and partly to the muscles supplied by this nerve being paralysed.

The *prognosis* and *diagnosis* call for no special remark, and the *treatment* must be upon general principles.

Glossoplegia.—Paralysis of the tongue.

Glossy Skin. *See* Nerves, Multiple Peripheral Neuritis.

Glottis. *See* Asphyxia (*Causes, Œdema Glottidis*); Bronchi, Bronchial Glands (*Morbid Anatomy, Pressure*); Hiccough; Larynx, Laryngismus Stridulus; Larynx, Acute and Chronic Inflammations (*Œdematous Laryngitis*); Mumps (*Complications, Œdema Glottidis*); Physiology, Respiration (*Voice, Larynx, Anatomy and Physiology*); Tabes Dorsalis (*Symptomatology, Laryngeal Crises*); Teeth (*Alveolar Abscess, Sequelæ, Œdema and Spasm of Glottis*); Thymus Gland (*Enlargement, Mechanical Effects*).

Gloves, Rubber.—Rubber gloves are much used in surgery for the carrying out of the principles of asepsis; they interfere slightly with the sense of touch, and various substitutes

have been suggested, such as a 4 per cent solution of gutta-percha in benzene or in acetone applied as a thin coating to the hands of the operator and the skin of the patient (in the area of operation).

Glucanth.—An excipient used in the making of pills; it contains tragacanth, glycerine, water, and syrup of glucose.

Glucosamine.—A sugar-like substance containing nitrogen ($C_6H_{11}NH_2O_5$); it occurs in mucin and chondroitin. *See* PHYSIOLOGY, TISSUES (*Mucin, Cartilage*).

Glucose.—The *glucoses* are sugars containing 6 atoms of carbon ($C_6H_{12}O_6$) and they are divided into the aldoses and ketoses; ordinary glucose or dextrose or grape-sugar is an aldose, and fructose or lævulose or fruit-sugar is a ketose. *See also* BLOOD (*Special Methods of Examination, for Glucose*); PHYSIOLOGY, TISSUES (*Carbohydrates*); PHYSIOLOGY, BLOOD (*Plasma and Serum, Contents*); PHYSIOLOGY, FOOD AND DIGESTION (*Food Stuffs yielding Energy*); POST-MORTEM METHODS (*Media, Bacteriological, Glucose Agar*).

Glucosides.—Vegetable substances which break up into glucose and other things (*e.g.* alcohols and aldehydes) when acted on by ferments or acids; examples of glucosides are found in chinovin (in cinchona bark), in salicin (in salix bark), and in scillitoxin (in squill bulbs).

Glue-Making.—Glue is made from hides, hoofs, horns, bones, etc., the residue of the process, which is called "scutch," gives off a very bad smell; glue-making is therefore regarded as an "offensive trade," and the "scutch" must not be allowed to accumulate but be removed at once (48 hours is the limit by law in England and Wales, 1875).

Glusidum. *See* SACCHARIN.

Gluteal Artery. *See* ANEURYSM (*Lower Limb, Gluteal Artery*); ARTERIES, LIGATURE OF (*Wounds, Operation of Ligature*).

Gluteal Nerve. *See* SPINE, SURGICAL AFFECTIONS (*Injuries of Sacral Plexus*).

Gluten.—A tenacious matter obtained from flour after the separation of the starch; it is said to be a mixture of vegetable fibrin, casein, and glutin (an albuminous substance).

Gluten Bread. *See* DIABETES MELLITUS (*Treatment, Diet*); INVALID FEEDING (*Cookery in Diabetes, Gluten Bread, Flour or Farina*).

Glutol.—An antiseptic made of gelatin and formic aldehyde, used as a dusting powder; glutoform.

Glycæmia.—The presence of sugar in the blood.

Glycerin. *See also* LABOUR, OPERATIONS (*Induction of Premature Labour*).—A trihydric alcohol ($C_3H_5(OH)_3$) manufactured by decomposing fats with alkalies or superheated steam. It is miscible with water and alcohol in all proportions, but not with ether and chloroform. *Dose*—1-2 ʒ. *Preparations*—(1) Glycerinum Acidi Borici, known as Boroglyceride; (2) G. Acidi Carbolici, 1 of phenol to 5 of glycerin; (3) G. Acidi Tannici, 1 to 5; (4) G. Aluminis, 1 to 6; (5) G. Amyli, 1 to 8; (6) G. Boracis, 1 to 6; (7) G. Pepsini, 5 grs. of pepsin in each ʒ. *Dose*—1-2 ʒ; (8) G. Plumbi Subacetatis; (9) G. Tragacanthæ; (10) Suppositoria Glycerini.

On account of its excellent solvent properties and its demulcent action glycerine is largely employed as a medium for the application of drugs to the mouth, throat, and skin. It is used alone for chapped hands and slight excoriations in any part of the body. It is absorbed when rubbed into the skin, and is thus a convenient vehicle to bring about the local action of anodynes and other remedies. By the mouth it is very frequently prescribed as a vehicle and flavouring agent. It also has a slightly laxative effect. Administered per rectum, either as a suppository or simply by injection of 1-2 ʒ, it induces a prompt and satisfactory action of the bowels, and it is therefore used in the treatment of some forms of constipation.

Glycerophosphates.—The glycerophosphate of iron (dose, 1 to 5 grains) and the glycerophosphate of lime (dose, 3 to 10 grains) are non-official preparations which are often used in the treatment of nerve-tire; they may be combined with formates.

Glyceryl.—The radicle (C_3H_5) of glycerin; it is sometimes written Gl, but is thus rendered likely to be confounded with Gl the symbol for Glucinum or Beryllium. *See* GLONOIN OIL.

Glycocholic Acid. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Intestinal Digestion, Bile Acids*).

Glycocoll.—Amido-acetic acid. *See* PHYSIOLOGY, EXCRETION (*Hippuric Acid, Formation*).

Glycoformal.—A disinfectant consisting of glycerine and a solution of formic aldehyde.

Glycogelatin.—A substance (consisting of gelatin, glycerin, and orange-flower water, coloured with carmine) used as a basis for throat lozenges. *See* GELATIN; PRESCRIBING; etc.

Glycogen.—A carbohydrate, $x(C_6H_{10}O_5)$, found in the liver, in the muscles, and other tissues, in the embryo, the placenta, etc., which

has the property of breaking up under the action of ferments (enzymes) and acids into other carbohydrates (*e.g.* glucose). *See* Blood (*Special Examination, for Glycogen*); Liver, Physiology of (*Regulation of Supply of Sugar*); Physiology, Tissues (*Chemistry of Muscle*); Physiology, Food and Digestion (*Fate of Food Absorbed, Liver, Glycogenic Function*).

Glycogen Reaction.—The occurrence of iodophilia in leucocytes is of considerable practical diagnostic importance. The reaction can be demonstrated very simply, using iodine, 1, potassium iodide, 3, water, 100, with enough gum arabic added to make a syrupy solution. No fixation is required, the dried film being mounted on a slide with a drop of the reagent, and examined after a minute or two. Extracellular brown-stained masses are of no diagnostic moment. Normally the leucocytes are stained pale yellow; when the glycogen reaction is present some of them take on a deep brown colour or show brownish red granular masses scattered through the protoplasm. The glycogen reaction is got in bad anæmia, in pneumonia, and in other acute diseases, but is particularly valuable as a diagnostic of the occurrence of suppuration.

Glycolysis.—The decomposition of glucose as accomplished by certain organs and tissues of the body (*e.g.* pancreas).

Glycoproteids.—Proteids linked with sugar-like substances (*e.g.* glucosamine) to form compounds, such as mucin.

Glycosal.—An antiseptic and antirheumatic drug, being a glycerin ester of monosalicylic acid.

Glycosuria.

See also Adrenal Glands (*Physiology*); Anæsthesia, General Physiology of (*Glycosuria after Chloroform*); Brain, Tumours of (*Localising Symptoms, Urine*); Children, Clinical Examination of (*Urinary System, Glycosuria*); Diabetes Mellitus; Digestion and Metabolism (*Absorption and Metabolism of Carbohydrates*); Gout (*Irregular, Renal System*); Influenza (*Effects on Different Systems, Kidneys*); Invalid Feeding (*Cookery in Diabetes*); Jaundice (*Phosphorus Poisoning, Symptoms*); Liver, Diseases of (*Signs of Cirrhosis, Urine*); Milk (*Therapeutic Uses*); Obesity (*Pathological Relations, Glycosuria*); Pancreas, Physiology of; Pancreas, Diseases of (*Pancreatitis, Cysts, Malignant Disease, Lithiasis*); Physiology, Food and Digestion (*Liver in Relation to Absorbed Food*); Pruritus; Puerperium, Physiology (*Excretory System, "Physiological Diabetes"*); Rabies (*Period of Excitement, Sugar in Urine*); Rheumatism, Rheumatoid Arthritis (*Clinical Characters, Subacute and Chronic, Urine*); Stomach and Duodenum, Diseases of (*General Symptomatology, Urinary*); Stomach and Duodenum, Diseases of (*Special Symptoms of Gastric Ulcer*); Thyroid Gland, Medical (*Exophthalmic Goitre, Symptoms, Urinary*); Urine, Pathological Changes in (*Sugars*); Whooping-Cough (*Complications, Urinary System*).

The term glycosuria is applied to that condition in which grape sugar is found *temporarily* in the urine. Thirst and diuresis, and other diabetic symptoms, are usually absent or very slight. Many authors would apply the term diabetes mellitus to all cases in which grape sugar is *permanently* present in the urine (in quantities sufficient to give a reaction to the ordinary clinical tests for sugar). Cases in which a small quantity of sugar is permanently present in the urine, whilst other symptoms are slight or absent, are, however, sometimes described by the term chronic glycosuria; but it appears better to describe such cases as mild forms of diabetes mellitus.

A temporary glycosuria is not usually followed by diabetes mellitus at a later period; but sometimes, after disappearing for a time, the glycosuria recurs, and occasionally becomes permanent, and the condition develops into one of true diabetes mellitus many months or several years after the glycosuria was first detected.

The following are the chief forms of glycosuria:—

1. Alimentary Glycosuria.—In the normal condition the power of sugar destruction of the body is not unlimited, and a very large quantity of saccharine food causes a small amount of sugar to appear in the urine. The various forms of sugar differ in their power of producing alimentary glycosuria. The following are the quantities of several forms of sugar which will produce glycosuria in healthy persons (v. Noorden): milk sugar over 120 grms., cane sugar 150-200 grms., fruit sugar about 200 grms., grape sugar 180 to 250 grms. (the sugar should be given in one dose, on an empty stomach before breakfast). The power of assimilation for starch appears to be unlimited in health. The power of sugar assimilation is sometimes slightly diminished in apparently healthy persons, and is often much reduced in various pathological conditions (as, for example, Graves' disease, cirrhosis of the liver, many fevers, chronic alcoholism, obesity).

In order to detect a diminution of the power of sugar destruction (alimentary glycosuria) in any individual, Naunyn recommends the follow-

ing test:—Two hours after a breakfast of coffee and milk ($\frac{1}{4}$ litre), with 80-100 grms. of bread, 100 grms. of grape sugar are given. If distinct glycosuria occurs, then the power of assimilation for sugar is diminished.

Some observers are of opinion that alimentary glycosuria (or *glycosuria e saccharo*) is simply the mildest form of that morbid condition which reaches an advanced state in spontaneous glycosuria (*glycosuria ex amylo*) or diabetes mellitus. Whether this view be correct or not, it has been found occasionally that cases of diminished sugar destruction or alimentary glycosuria (detected by the 100 gramme glucose test as above described) have, in course of time, developed into diabetes mellitus.

Thus v. Noorden found that in 4 out of 15 obese persons, temporary alimentary glycosuria was produced by 100 grammes of glucose. Of these four individuals two became diabetic several years later.

In cases of chronic alcoholism from excessive beer-drinking, Strümpell has drawn attention to the frequency of alimentary glycosuria or diminished power of sugar destruction; and he believes that there is no sharp line between this condition and the true diabetes which is sometimes met with in alcoholic subjects.

In traumatic neurosis or neurasthenia there is often a diminished power of sugar destruction; also Ebstein and Asher have shown that traumatic neurasthenia is sometimes followed by diabetes mellitus.

These facts are sufficient to show that the detection of a diminished power of sugar destruction (or alimentary glycosuria) by means of the 100 gramme sugar test, may be of some practical value with respect to the prophylaxis of diabetes mellitus. Thus, for example, in cases of great obesity, in chronic alcoholism (especially from beer-drinking), in traumatic neurasthenia, and in individuals having a family history of diabetes, if the 100 gramme sugar test shows that there is a diminished power of sugar destruction, it is possible that, by a suitably restricted diet, the development of diabetes mellitus might be prevented.

In such cases sugar and certain articles very rich in starch should be forbidden; also sweet wines and beer should not be taken.

In the urine examinations for life assurance sometimes a trace of sugar is detected, though there is no other evidence of disease. In such cases not infrequently the sugar disappears rapidly, and the urine is normal next day. Here also the 100 gramme sugar test would be of value as a means of showing whether there was any diminution in the power of sugar destruction, and of course, if a positive result were obtained, a modified diet would be indicated.

2. Puerperal Glycosuria (*Lactosuria*).—It has long been known that during the puerperal state a substance occurs temporarily in the urine which reduces Fehling's solution. It has been shown that this substance is lactose in small quantity. It is said to be most abundant on the fourth or fifth day of the puerperium, and it appears to be connected with engorgement of the breasts.

3. Glycosuria produced by various Drugs and Poisons.—(*a*) *Phloridzin diabetes.*—It has been shown by v. Mering that phloridzin, given hypodermically or by the mouth, produces glycosuria with increase in the quantity and specific gravity of the urine, both in man and in animals. These symptoms disappear when the drug is discontinued. According to some observers there is no excess of sugar in the blood, and the glycosuria is thought to be due to the action of the drug on the kidneys.

(*b*) Other drugs and poisons *sometimes* produce slight glycosuria if given in very large doses; but this result is by no means constantly obtained. Glycosuria has *occasionally* been observed after toxic doses of the following drugs: opium, morphia, chloral hydrate, prussic acid, mineral acids, mercurial salts, arsenic, phosphorus, uranium salts, curare, orthonitro-phenyl-propiolic acid, caffein, theobromine, diuretin, caffein, sulphonic acid, ether and chloroform narcosis, coal gas and carbon monoxide poisoning.

4. Glycosuria associated with various Diseases.—In a number of varied diseased conditions a slight and temporary glycosuria is met with occasionally, whilst in similar conditions it is frequently absent. The following are some of the conditions in which glycosuria has been sometimes noted: injuries to the head, concussion, fracture of the skull, cerebral hæmorrhage, meningitis, brain tumours (very rare except in tumours of the pituitary body, see article on "Diabetes"), Graves' disease, locomotor ataxia (very rare), disseminated sclerosis (very rare), mental diseases (chiefly melancholia), cirrhosis of the liver, obesity, gout; occasionally after acute fevers, such as typhoid, scarlet fever, measles, diphtheria, cholera; occasionally temporary glycosuria occurs during pregnancy. Glycosuria or diabetes is often associated with acromegaly.

(With respect to the relation of gout, syphilis, arterio-sclerosis, and obesity to diabetes and glycosuria, see article on "Diabetes." In this article the forms of glycosuria after removal of the pancreas and after experimental lesions of the nervous system are also described.)

5. Pentosuria.—Recently a number of cases have been recorded in which the urine has reduced Fehling's solution, and the reducing body has been shown to be pentose—a form of sugar containing five atoms of carbon or a multiple thereof in the molecule. No definite symptoms have been associated with the presence of pentose in the urine.

Detection of Glycosuria.—As considerable care is sometimes necessary in distinguishing

between small quantities of sugar and other substances occasionally found in the urine, a few words on the clinical tests may be of service. There are numerous tests for grape sugar in the urine, but the three most valuable are Fehling's solution, phenyl-hydrazin, and fermentation. By the employment of these three tests in the following manner and order, as a rule, grape sugar can be easily detected with certainty, even when only a trace is present.

Fehling's solution is the most convenient test. It is important to boil the test solution first: if reddish yellow oxide of copper be thrown down, or if a greenish turbidity be produced, the Fehling's solution has decomposed and a new specimen is necessary. If no change occur the solution is good, and a little urine may now be added, and the mixture boiled. It is important not to add too much urine (never more than the quantity of Fehling's solution employed). Also, the mixture should not be boiled too long. (If these precautions be not taken, a slight turbidity may occur when the urine contains a great excess of uric acid or urates.) If on testing with Fehling's solution *no* reaction be obtained, we may safely conclude that the urine is free from sugar in the clinical sense, and *no further testing* is necessary.

If an abundant reddish or yellowish precipitate (copper oxide) occur on boiling the urine with Fehling's solution, the presence of grape sugar may be assumed, providing the precautions mentioned have been taken, and providing other signs of diabetes mellitus be present, such as increased quantity and specific gravity of the urine, diuresis, thirst, etc. But the difficulty arises chiefly when diabetic symptoms are absent, and when the only evidence of disease is the presence of a substance in the urine which reduces Fehling's solution. Often the reduction is slight: it may consist only of a greenish turbidity, occurring when the test-tube cools. Sometimes this slight change is due to sugar, sometimes to other substances which occasionally occur in the urine, and which reduce Fehling's solution, such as glycuronic acid, alkapton, uric acid and urates in great excess, lactose, pentose, etc. Hence some confirmatory test must be employed before a definite opinion can be given. The best two confirmatory tests are phenyl-hydrazin and fermentation.

The following simple method of performing the *phenyl-hydrazin test* is of great service, and if a *negative* result be obtained we can safely state that the substance which caused the reduction of Fehling's solution was not grape sugar. A test-tube of ordinary size is filled for *half an inch* with hydrochlorate of phenyl-hydrazin (in powder); then acetate of soda (in powder or fine crystals) is added for another *half-inch.* The test-tube is half-filled with urine and heated directly over a spirit lamp (*without* the use of a water bath). For two minutes the urine is kept boiling. The tube is then placed in the test stand, and twenty or thirty minutes later, if grape sugar be present, the deposit at the bottom of the test-tube is found, on microscopical examination, to contain needle-shaped crystals of a bright yellow colour. Often the crystals are arranged in sheaves or rosettes. If these crystals cannot be detected, the urine is free from grape sugar in the clinical sense, and no further testing is necessary. An abundant deposit of the crystals in the test-tube is in all probability due to grape sugar. But lactose produces yellow globules with short spines, and glycuronic acid small yellow crystals. Hence when a positive result is obtained with the phenyl-hydrazin test it is always best to employ the fermentation test, which is the most reliable method.

The *fermentation test* may be conveniently carried out in the following manner:—Two ordinary test-tubes of equal size are employed. The same quantity of German yeast (weighed) is placed in the bottom of each (about $\frac{1}{10}$ of the tube is filled with the yeast). One test-tube is filled with normal urine, the other with the suspected urine. The mouth of each test-tube is closed with an india-rubber stopper, perforated with a short piece of glass tubing bent twice at right angles. The two test-tubes are inverted and kept side by side in a warm place, being supported in a glass tumbler. In twenty-four hours, at the top of the test-tube containing the normal urine with yeast, a small bubble of gas will have collected. (This gas is given off by the yeast itself.) At the top of the tube containing the suspected urine with yeast, if grape sugar be present, a greater quantity of gas will have collected. The gas may occupy one-quarter, or one-half, or the whole of the tube, according to the quantity of sugar present. Any excess of gas in the tube containing the suspected urine, beyond the small bubble of gas in the tube containing the normal urine, will indicate grape sugar or other fermentable sugar with certainty.

Glycoxylic Acid. *See* Liver, Physiology of (*Allantoin*).

Glycozone.—A proprietary preparation, believed to act as an antiseptic by reason of the ozone which it develops.

Glycuronic Acid. *See* Physiology, Tissues (*Cartilage, Decomposition of Chondroitin*); Urine, Pathological Changes in (*Aromatic Substances, Glycuronates*).

Glycyrrhizae Radix. *See* Liquorice.

Gmelin's Test. *See* Urine, Pathological Changes in (*Bile Pigment*).

Gnat Fever. *See* Malaria (*Synonyms*).

Gnathalgia.—Pain in the jaw, usually of a neuralgic type.

Gnathion.—A projecting point on the anterior inferior margin of the lower jaw in the middle line.

Gnathocephalus.—A teratological type in which the head consists almost solely of the jaws.

Gnathoschisis.—Cleft palate.

Gnathospasmus.—Trismus on tonic contraction of the muscles which clench the jaws. *See* TETANUS.

Gnats. *See* MYIASIS (*Insect Stings and Bites*); STINGING INSECTS (*Gnats*).

Goa Powder. *See* CHRYSAROBINUM.

Goats. *See* ANTHRAX (*In Animals, Goats*); INFANT FEEDING (*Milk of Animals other than the Cow*); MILK (*Physiological, Goat's Milk*).

"Godfrey's Cordial." *See* TOXICOLOGY (*Opium and Morphine*).

Goitre. *See* THYROID GLAND, MEDICAL (*Goitre and Exophthalmic Goitre*); THYROID GLAND, SURGERY OF (*Goitre and Exophthalmic Goitre*). *See also* ANÆSTHESIA (*Chloroform in Goitrous Subjects, Attitude*); CORNEA (*Ulceration from exposure, Exophthalmic Goitre*); HÆMATOPORPHYRINURIA (*in Exophthalmic Goitre*); NOSE, NASAL NEUROSES (*Cause of Symptoms of Goitre*); WATER (*Diseases produced by*).

Gold Cure.—A secret method of treating alcoholism; Keeley cure; the medicine used is said to contain gold.

Gold-Filling. *See* TEETH (*Mechanical Dentistry*).

Golgi, Organs of. *See* PHYSIOLOGY, SENSES (*Muscle Sense, Structure of Muscle*).

Golgi, Staining Method of. *See* PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum, Arrangement of Neurons*).

Gonagra.—Gout in the knee-joint. *See* GOUT.

Gonalgia.—Pain in the knee, often neuralgic in type.

Gonarthritis.—Inflammation or gout in the knee.

Gonecystitis.—Inflammation of the seminal vesicles.

Gonion.—The angle of the lower jaw and the region round it.

Gonococcus. *See* GONORRHŒAL INFECTION; MICRO-ORGANISMS; URETHRA, DISEASES OF (*Gonorrhœa, Pathology*).

Gonorrhœa. *See* URETHRA, DISEASES OF (*Gonorrhœa*). *See also* ANKLE-JOINT, REGION OF, DISEASES (*Gonorrhœal Affections*); BURSÆ, INJURIES AND DISEASES OF (*Gonorrhœal Bursitis*); CONJUNCTIVA, DISEASES OF (*Purulent Ophthalmia*); GONORRHŒAL INFECTION; HIP-JOINT, DISEASES OF (*Gonorrhœa*); IRIS AND CILIARY BODIES (*Inflammatory Conditions, Gonorrhœal Iritis*); JOINTS, DISEASES OF (*Gonorrhœal Affections*); KNEE-JOINT, DISEASES OF (*Pyogenic, Gonorrhœal Affections*); MENINGES OF THE CEREBRUM (*Purulent Meningitis, Etiology*); MICRO-ORGANISMS (*Gonococci*); MYOSITIS (*Secondary, Gonorrhœal*); NAILS, AFFECTIONS OF (*Intoxications, Gonorrhœa*); NERVES, MULTIPLE PERIPHERAL NEURITIS (*General Etiology*); NOSE, EXAMINATION OF (*Character of Secretion, Bacteriology*); PERITONEUM (*Pathology, Bacteriology*); POST-MORTEM METHODS (*Bacteriological Investigations, Gonorrhœa*); PURPURA (*Symptomatic, Infections*); PYURIA; SCROTUM AND TESTICLE, DISEASES OF (*Eruptions on Scrotum, Gonorrhœal Warts*); SPERMATORRHŒA; SUPPURATION (*Etiology*); URINE, BACTERIA IN (*Gonococcus*); UTERUS, INFLAMMATIONS (*Metritis, Endometritis, etc.*); VAGINA, DISORDERS OF (*Inflammations, Gonorrhœal Vaginitis*); VULVA, DISEASES OF (*Inflammation, Condylomata*); WRIST-JOINT, DISEASES OF (*Gonorrhœal Arthritis*).

Gonorrhœal Infection.—Although the special inflammations resulting from an attack of gonorrhœa have been considered under their appropriate headings, it will be convenient to summarise the effects produced by gonorrhœal infection in the female.

In the acute form the vulva, vulvo-vaginal glands, and urethra are involved, accompanied by swelling of the inguinal glands and even abscess formation. The vagina is stated by some authorities to be rarely affected, but this depends entirely on the character of the epithelial lining. In children the vagina is affected. In adults where connection has occurred frequently, the vaginal walls offer more resistance to the action of the poison. A secondary vaginitis is not infrequently produced through the discharge from the cervix uteri, causing desquamation of the superficial layers of the vaginal epithelium. Œdema of the labia is a most important sign, and is frequently accompanied by superficial ulceration. The urethral orifice is inflamed, and on pressure on the urethra with the finger introduced into the vagina a purulent discharge exudes. When the vagina is involved there is marked congestion, and may be hæmorrhages, and the surface is bathed with a thin purulent discharge.

The distinction between a gonorrhœal attack and one due to other causes is chiefly the degree of inflammation, and previous to the discovery of the gonococcus the severity alone was relied upon to settle the diagnosis. Now,

however, the discharge should be examined microscopically to discover the presence or absence of the special micro-organism. In acute cases the cervical mucosa is attacked, and a copious purulent discharge flows from the cervical canal. The systematic examination of the discharges in cases of gonorrhœa has demonstrated the important influence of the cervical discharge, not only in acute, but also in chronic gonorrhœa. In the latter group of cases the cervical discharge remains infective for a lengthened period. The gonorrhœal poison tends to persist for a long time in certain situations, viz. the cervical mucosa, the ducts of the vulvo-vaginal glands (glands of Bartholin), and lastly, the urethra.

Where suitable treatment has been employed the disease may be cured before it has spread farther, and the more serious complications averted. If, however, douching, the incautious use of the sound or other instrument, has caused an upward extension of the poison, the body of the uterus, the ovaries and Fallopian tubes, the pelvic peritoneum, and cellular tissue become in turn involved in the disease. Owing to the changes produced in the ovaries and Fallopian tubes sterility is a frequent consequence. Well-marked examples of what has been termed acute ascending gonorrhœa are found in puerperal cases, where infection has occurred late in pregnancy, and where the changes in the genital tract produced as a result of the delivery of the child favour the upward extension of the gonorrhœal poison. During pregnancy, owing to increased secretion of mucus and epithelial desquamation, the vagina is more liable to be affected.

The severity of the attack in acute cases prompts the sufferers to seek medical relief. In the subacute and chronic cases, where the symptoms are slight, the disease is allowed to progress untreated, unless some further complication arises. There is further the difficulty in persuading the class of patients who suffer from gonorrhœa to continue treatment until they are completely cured. Such patients with a gonorrhœal discharge from the cervix uteri continue to be sources of infection to those with whom they come in contact.

When a case of endo-cervicitis comes for treatment, it is well to examine the discharge for gonococci, for, as already mentioned, the passage of the sound or other instrument favours the upward extension of the gonorrhœal poison. It should be added that the periodic congestion resulting from the menstrual functions tends to keep up or aggravate the cervical catarrh. There is considerable difficulty in estimating the proportion of cases of salpingo-oöphoritis which are due to gonorrhœa. Statistics have been published by several German writers with a view to determining the exact proportion. The statement that $\frac{1}{5}$th of the cases is due to gonorrhœal infection is probably correct. Septic infection, the result of a confinement, a miscarriage, or "persistent local treatment!" to the cervix uteri, and tuberculous infection, are answerable for the remainder.

The clinical features of the cases of ascending gonorrhœa, with implication of the Fallopian tubes and ovaries, will be best understood by describing a typical example :—

A healthy young woman has married a man who had contracted gonorrhœa six months previously, and who believed he was cured. Three or four days after marriage his wife complains of pain and soreness on passing water, accompanied by vaginal discharge. The intensity of the initial manifestations may vary considerably, but the history of definite local signs and symptoms can usually be obtained. The vaginal discharge persists long after the dysuria has disappeared. Menstruation, which before marriage had been regular and almost painless, soon becomes irregular and preceded by considerable pain. The pain commences gradually in the intermenstrual periods, and is aggravated by standing or exertion. It is felt not only in the hypogastric and iliac regions, but also in the sacral region, and extending down the thighs. The pain may or may not be relieved by the menstrual flow.

The patient soon begins to lose flesh, and is unable to do any work, and, as the severity of the symptoms increases, may be entirely confined to bed. She becomes a chronic invalid, dragging on a weary existence, never free from aches and pains. During the progress of the illness the pain is liable to acute exacerbations, accompanied by elevation of temperature, thirst, nausea, and actual vomiting. Such attacks are frequently termed by patients "inflammation of the bowels," but are, in reality, due to pelvic peritonitis, spreading from the Fallopian tube, or in the severe cases actual leakage of pus through the abdominal ostium before it has become occluded. Sterility develops as a consequence of the tubal disease.

On *abdominal examination* either no swelling may be detected, or a swelling in the iliac regions, tympanitic on percussion due to adherent intestine, which may in bad cases form a roof to the pelvis, thus rendering an abdominal operation very difficult. Where large collections of pus have formed in the tubes these may be palpated per abdomen.

Vaginal examination reveals a mass situated on one or both sides of the uterus posteriorly. If unilateral, the uterus is pushed towards the sound side, and will probably be movable with the mass. The vaginal roof on the affected side is depressed. If bilateral, the masses, when of large size, may interfere with the mobility of the uterus, that organ being fixed in the centre of a bilateral swelling. The

vaginal roof on both sides is depressed. The masses in the pelvis are either the tubes and ovaries matted together by adhesions, or definite purulent collections in the tubes or ovaries, or in both.

Where definite purulent collections exist, the pus must be evacuated. The method of doing this need not be discussed here. The treatment of the cases where no purulent collection exists has led to much controversy. The results of operation are disappointing; pain frequently persists; whereas the expectant treatment by rest and suitable remedies has given fairly good results, and should always be tried before an operation is recommended. Complete recovery may follow when gonorrhœal salpingitis has been properly treated. It is erroneous to suppose, as some writers do, that all cases require operation.

A word may be said concerning the sterility which results from gonorrhœal infection. There may be absolute sterility, but more frequently the sterility is relative (the "one-child sterility" of Sänger). The phenomenal sterility of prostitutes is probably due in part to the presence of adhesions fixing the Fallopian tubes and ovaries, and, further, to atrophic changes occurring in these organs as a result of preceding disease. Many prostitutes have had one child, and are afterwards sterile. The birth of the child may have been the cause of their adopting this mode of life, or it may have favoured the upward spread of the gonorrhœal poison.

The diagnosis of gonorrhœal cases, where the history is indefinite or unreliable, is always difficult apart from bacteriological tests. If, however, a patient who shortly after marriage develops symptoms as detailed in the illustrative example, and who has not had any local treatment or vaginal examination, nor any miscarriage or confinement, and in whom tubercle can be excluded, the cause is probably gonorrhœa.

"Latent Gonorrhœa."—Many writers have persisted in stating that an attack of gonorrhœa cannot be completely cured, and although apparently cured, tends to recur after alcoholic excess or sexual indulgence.

Where treatment has been ineffectual or neglected, the discharge may persist, but with suitable remedies the disease can be thoroughly eradicated. Ever and anon cases occur which are capable of producing gonorrhœal infection for two or more years. I know of no case definitely authenticated in which the pus contained gonococci after four years.

In the majority of the very chronic cases reported the proof of the presence of gonococci is incomplete.

Noeggerath,[1] whose statements are much exaggerated, insisted on the importance of what he termed "latent gonorrhœa." He believed that a man is never completely cured of an attack of gonorrhœa, and that that man is certain to infect his wife.

It is further supposed that in cases of so-called "latent gonorrhœa" indulgence in alcohol or marital intercourse brings to life certain gonococci which are lying in a dormant state.

If an attack of gonorrhœa in the male remains uncured, the affection is increased by indulgence in alcohol or by sexual intercourse. This, however, is a case of chronic or neglected gonorrhœa, not one of so-called latent gonorrhœa.

After a severe attack, even when the disease has been cured, a discharge, purulent or mucoid, may result after alcoholic or sexual indulgence. In such cases, however, no gonococci can be demonstrated in the pus.

Where a patient has had several attacks of gonorrhœa the urethra becomes structurally altered, and although these attacks confer a certain immunity as regards the action of the gonococcus, the urethral mucosa becomes unduly sensitive to the action of other pus organisms. Any increased congestion or irritation may give rise to a purulent urethritis. No gonococci are found in the pus.

The great advantage which has accrued from Noeggerath's work is that the medical profession, and through it the public, have been educated to the importance of having an attack of gonorrhœa completely cured. Further, the suffering and misery which attends gonorrhœal infection in the female has been demonstrated by other workers, and patients are now treated in the early stages, and the diseased cured before important internal organs are involved.

The suggestion of the term "latent gonorrhœa" has led to a deal of what may be termed speculative pathology, which has been ridiculed by those opposed to the influence of micro-organisms in disease. It would be well to discontinue this term, as such cases are better named uncured, neglected, or chronic gonorrhœa.

The Character of the Infection which causes the Complications and Sequelæ of Gonorrhœa.—This is a difficult question to decide, and in the present state of our knowledge we are unable to arrive at definite conclusions.

The possibilities may be briefly stated as follows:—

(1) Pure gonorrhœal infection, the result of the invasion of gonococci alone.

(2) Mixed infection, the result of the invasion of pus organisms, which have developed in the mucous surface originally attacked by the gonococci.

(3) Secondary infection, where the gonococci produce the complication, followed later by the

[1] *Die latente Gonorrhœa im weiblichen Geschlecht.* Bonn, 1872.

entrance of pus organisms, both existing together until gonococci die or are overgrown by the pus cocci.

(4) Toxic infection—the complications may be the result of the absorption of toxic products produced by the gonococci or the pus organisms.

Under one or more of these headings the complications and sequelæ of gonorrhœa may be explained.

It is probable that a certain amount of toxic infection is present in every case.

From the writer's own observations he is led to conclude that the effect of the action of the gonococcus on the particular soil attacked is to convert that soil into a suitable medium for the growth and development of pus organisms.

If the invasion of pus organisms be great we may have various complications developed quite apart from the action of the gonococcus.

In the consideration of this subject we may be led astray if, after constantly finding the same collection of organisms in certain lesions, we fix on one species as specific, and neglect the others as contaminations.

The majority of the complications in the female are due to a mixed infection or to a secondary infection. The cases of rapid upward spreading of the disease, attended by the formation of pyosalpinx, are in all probability examples of secondary infection. The more chronic cases are rather examples of mixed infection.

The more efficient drainage for the female discharges renders the occurrence of marked toxic phenomena less frequent. Where, however, the outflow is obstructed, as in certain cases of pyosalpinx and ovarian abscess, then toxic phenomena do occur. The presence of such phenomena, however depends entirely on the virulence of the purulent contents, as many cases of pyosalpinx and ovarian abscess are unattended by much systemic disturbance, not even elevation of temperature.

Although it is stated that articular complications are relatively rare in women, it will be found that in the majority of cases a history of joint pains can be elicited. The occurrence of effusion into one or more joints is not uncommon, and ankylosis may develop as a consequence. More frequently, however, the joint affections are not severe, consisting in pain on movement accompanied by little or no effusion. The possibility of gonorrhœal infection as a cause of synovitis in women should always be remembered. In the lower extremity the knee and ankle joints are most commonly affected. In the upper extremity the wrist-joint is selected.

Treatment.—This is discussed under the Complications of Gonorrhœa, and only a short summary need be given here.

In acute cases rest in bed should be advised, and in addition the avoidance of injections, which undoubtedly are very harmful.

The writer does not recommend douching even in chronic cases, as in both the treatment can be more effectually and thoroughly done by swabbing out the vagina through a speculum with strong silver nitrate or strong carbolic acid solution. A similar application should be made to the endo-cervix. It is not necessary nor advisable to make application to the endometrium, for practical experience teaches that gonorrhœal endometritis proceeds to a natural cure. For this reason also curetting should not be recommended, as it is not free from danger. In acute cases an anæsthetic may be required owing to the pain and difficulty in introducing a speculum. If, however, this treatment by swabbing out the vagina be properly carried out the onset of the disease may be checked.

Too frequently the patients have used douches, or have been advised to use them, with the result that the upward extension of the disease has been increased.

Now that gonorrhœa has been proved to be due to a microbe whose effects are at first purely local, involving mucous membranes, the rational treatment consists in an early destruction of this microbe. Moreover, in the female it is easy to expose the affected area and to apply the germicide directly on to the diseased surface; whereas in the male the difficulty is greater even if aided by the urethroscope. Douching should therefore be abolished in the treatment of gonorrhœa in the female, and the method of direct application of the germicide to the diseased area should be substituted.

For this purpose a germicide is required which has the power of soaking into the mucous surface. Nitrate of silver and strong carbolic acid possess this power, and are the best applications. These caustics should be applied with care at first twice weekly, then once a week and gradually discontinued.

The endocervix and vaginal walls should be seared and care taken that no excess of the fluid is allowed to remain in the vagina. No gauze or lint soaked in the fluid should ever be left in the vagina, as sloughing of the vaginal mucosa may be produced.

A diaper of antiseptic wool and gauze should be worn to receive the discharges. It should be frequently changed, as local cleanliness is of the greatest importance.

If the immediate treatment of gonorrhœal infection was more efficiently employed, less would be heard of those serious forms of ascending gonorrhœal inflammation.

In pregnant women a thorough swabbing out of the vagina will prevent the upward spreading of the disease after the confinement, and will save the eyes of the child from infection at birth.

In addition to rest in bed and avoidance of douches the bowels should be kept freely open by saline aperients. Alcohol should be forbidden. For further details the reader is referred to the special articles "Vaginitis," "Vulvitis," etc.

Medico-legal Aspects of Gonorrhœal Infection.—The discovery of the gonococcus was at once hailed as of importance in medico-legal work, *e.g.* in cases of rape where gonococci were discovered in the vulvo-vaginal discharge of the victims when the accused suffered from gonorrhœa.

This is certainly an important corroboration; still bacteriological science is not yet sufficiently exact to warrant our making very definite statements. There are still very many important points requiring elucidation before we can reach that degree of accuracy required for expert evidence in a court of law.

A great controversy has existed for some time, and still exists, concerning the nature of the vulval discharges in infants and young children. Indeed, there occurrence has been used as a foundation for false charges of rape against innocent individuals. In such cases a careful examination should be made of the vulva, noting the presence or absence of excoriations, and in addition the discharge should be microscopically examined to determine the presence or absence of (*a*) spermatozoa, (*b*) gonococci. The chief difficulty surrounds the identity of the gonococci, for various Continental authorities have stated that these cases of vulvo-vaginitis in infants and young children are all due to gonorrhœa,—some being caused by direct infection, while others are due to infection from dirty towels, linen, etc.

In this relation it must be remembered that there is a popular superstition that sexual intercourse with a virgin will cure an attack of gonorrhœal urethritis.

A series of cases of vulvo-vaginitis in children were carefully examined by the writer of this article, with the result that although a few are gonorrhœal the great majority are not.

The reason why so many conflicting statements have appeared in papers written on this subject is that, as the writer believes, mistakes have been made over the identity of the organism present.

In the vulvo-vaginitis of infants and young children the discharge frequently contains an organism resembling closely in form and colour reactions the gonococcus, but differing in this important point, that it can be cultivated on gelatine, whereas the gonococcus does not grow on this medium.

It is this organism which has been mistaken for the gonococcus, and has led to the erroneous statements which have been made on the etiology of vulval discharges in children.

The identity of any micro-organism is not complete until the most reliable evidence of all, viz. the cultivation test, has been furnished. If then the purulent discharge be examined microscopically, and the organisms cultivated, a more correct view of the causation of this disease will be obtained. It will be found that a large number of the contributions on this question may be disregarded, as in the investigation of the subject the cultivation test was not employed.

In countries where prostitutes are systematically examined, bacteriological tests should be applied to the discharges before declaring that a woman is free from infection. The statistics furnished from various large Continental cities show how frequently gonorrhœa is found among women of this class. The bacteriological tests (microscopic and cultural) require to be oft repeated, many specimens of the discharge being examined, and at different periods, before a definite opinion is given.

Gonorol.—A preparation containing only certain constituents of sandal-wood oil, and said to act better in cases of gonorrhœa than the sandal-wood oil itself.

Gonosan.—A preparation highly spoken of in the treatment of gonorrhœa; it is a 20 per cent solution of the resins of the Polynesian kawa-kawa root in sandal-wood oil.

Goose-Cough.—The paroxysmal, wheezy cough, due to pressure on the recurrent laryngeal nerves, which occurs in cases of aneurysm of the thoracic aorta.

Goose-Skin. *See* CUTIS ANSERINA.

Gossypii Radicis Cortex.—The bark of the root of *Gossypium herbaceum* is used officially in India and the Colonies for the same purposes as ergot; there is a *Decoctum Gossypii Radicis Corticis* of which the dose is ½ to 2 fl. oz; and an *Extractum Gossypii Radicis Corticis Liquidum*, of which the dose is ½ to 1 fl. dr.

Gossypium.—Cotton-wool, consisting of the hairy appendages of the seeds of *Gossypium barbadense* and other species; "absorbent" cotton-wool has had the fatty matter removed, "non-absorbent" or ordinary cotton-wool has not; pyroxylin is made from cotton-wool by immersion in a mixture of sulphuric and nitric acids, and from it is obtained collodion (*q.v.*).

Goulard Extract.—Liquor Plumbi Subacetatis Fortis or strong solution of the subacetate of lead. *See* LEAD.

Goulard Lotion.—Liquor Plumbi Subacetatis Dilutus or Goulard Water. *See* LEAD.

Goundu.—A disease of the nose, common on the Gold Coast, in which, after an attack of yaws, two bony swellings appear one on each side of the root of the nose and cause great deformity of the face; they are due to osteoplastic periostitis; henpuye, anakhre, or dog-nose. *See* NOSE, CHRONIC INFECTIVE DISEASES (*Henpuye*).

Gout.

Fr. *Goutte*; Ger. *Gicht*.

See also Alcohol (*Clinical Uses, Gout*); Ankle-Joint, Region of, Diseases (*Gouty Arthritis*); Asthenopia (*Neurasthenic*); Asthma (*Treatment*); Balneology (*General Balneo-Therapeutics, Disorders of Nutrition and Elimination, Gout*); Bronchi, Bronchitis (*Acute, Etiology*); Bursæ, Injuries and Diseases of (*Bursitis, Gouty*); Deformities (*Hallux Valgus, Metatarsalgia*); Diabetes Mellitus (*Predisposing Causes*); Diathesis (*Gouty*); Ear, External, Diseases of (*Morbid Growths, Gouty Deposits*); Eczema (*Etiology and Classification, Gouty*); Guaiacum; Heart, Myocardium and Endocardium (*General Pathology, Etiology*); Hydropathy (*Subacute and Chronic Diseases*); Indigestion (*Treatment of Special Forms, Gouty*); Insanity, Etiology of (*Autotoxic*); Insanity, Nature and Symptoms (*Etiological Varieties*); Leucocytosis (*Toxic*); Meninges of the Cerebrum (*Purulent Meningitis, Etiology*); Menstruation and its Disorders (*Dysmenorrhœa, Causes*); Migraine (*Causes*); Nails, Affections of (*General Diseases, Gout, Reedy Nails*); Nephritis (*Renal Cirrhosis, Gout*); Nerves, Peripheral (*Neuritis, Causes*); Nerves, Multiple Peripheral Neuritis (*General Etiology*); Nerves, Neuralgia (*Etiology*); Obesity (*Pathological Relations*); Oxygen; Parotid Gland, Disorders of (*Inflammatory Conditions, Causes*); Penis, Surgical Affections of (*Cavernitis, Causes*); Pruritus (*Causation*); Pulse (*Frequency, Causes*); Rheumatism; Sclerotic, Diseases of (*Sclerotitis*); Scrotum and Testicle, Diseases of (*Epididymitis, Orchitis, Testalgia*); Stomach and Duodenum, Diseases of (*General Symptomatology, Gouty Dyspepsia*); Teeth (*Dental Caries, Saliva*); Teeth (*Diseases of Peridental Membrane, Causes*); Tongue (*Nervous Affections, Gouty*); Tonsils, Diseases of (*Acute Tonsillitis, Causes*); Toxicology (*Chronic Plumbism, Effect of Gout*); Trades, Dangerous (*Gout and Plumbism*); Ulceration (*Gouty*); Unconsciousness (*Autointoxications, Gout*); Urine, Pathological Changes in (*Elimination of Nitrogen in Gout*); Urticaria (*Causes*); Veins, Diseases of (*Gouty Phlebitis*).

Introduction.—It has been truly said that if a sprightly writer wished to make fun of the medical profession, nothing would give him a better opportunity for the exercise of his sarcasm than the extreme variety and the very opposite character of the regime which this, that, and the other medical authority have laid down for the dietetic treatment of the gouty state. Some recommend a diet mainly vegetarian in character, others find salvation in meat and hot water, and, apart from these two extremes, when we have regard to the numerous articles of food and drink in daily use, we find very few that are not as stringently forbidden by some writers as they are highly commended by others. The reason for this is readily found in our ignorance of the exact nature of the disease. After ages of inquiry we know gout only by the order and character of its phenomena, and have yet to learn its intimate nature or the special cause which produces it. We know so very few definitely established facts of importance in the etiology of the disease that a doubting practitioner may well be pardoned when he asks if we know any. But, in proportion as the facts are few, the hypotheses are numerous, and the too ready acceptance of conclusions, lacking confirmation in generally accepted facts, has been responsible in great measure for retarding our knowledge of the disease.

As we cannot yet give the physiological history of gout, we should adopt a pathology founded solely on facts unalloyed by hypotheses, and conformable to all that we know of the physiology of animal life. In the course of our inquiry as to the appropriate treatment it cannot fail to be remarked how much this treatment is dependent on the same general principles which guide us in other diseases, and how little consideration it admits for the special nature or character of gout.

There are two points that should ever be before those who work or write on this subject. All clinical experience teaches us that we are dealing with a constitutional disease whose natural history includes a liability to rapid and apparently inexplicable variations, often fortunately of the nature of amelioration or complete disappearance of symptoms. If these natural variations occur coincidently with the use of some supposed therapeutic measure, medicinal or otherwise, there is a risk of the observer wrongfully ascribing the benefit to the therapeutic agent employed, when it is in reality due to the self-righting power of nature. He becomes what Sir W. Roberts aptly termed the victim of misinterpreted sequences. Another fruitful source of error is the laboratory. Only

too frequently have measures which may have appeared on theoretical grounds to be applicable to the treatment of the disease succumbed before the searching light of practical experience. The fallacies in experimental laboratory work are so numerous that great care must be exercised in drawing any conclusions from this line of inquiry, and should the conclusions arrived at not be in harmony with the teachings of clinical experience they may well be abandoned.

A few words about the term *uric acid diathesis.* Under this term are generally included two conditions—gout and lithæmia—which have only one important point in common. In both the excretion of free uric acid in the urine plays an important part. The frequent association of these two conditions, either in the same individual or in different members of the same family, has not unnaturally led to the belief that there is an exceedingly close relationship between them, even to the extent of regarding uric acid calculi as gouty deposits in the urinary tract. While we must admit the probable existence of a very close relationship between the two conditions, they yet present sufficient points of difference in their appearances and results as to justify us considering them separately. The etiology and treatment of uric acid calculi will be considered later (see "Uric Acid"); for the present we are concerned with gout proper. The subject is admittedly a complex and difficult one, and while its adequate presentation involves frequent entry into unknown and purely speculative regions, I shall seek to indicate them clearly, so that we may avoid arriving at any conclusions not based on definitely ascertained and accepted facts.

Morbid Anatomy.—The characteristic anatomical changes in gout are dependent on the deposit of urate of soda in various parts of the body, notably in and around joint structures, and on secondary changes arising from this deposit. Much discussion has from time to time taken place as to whether degenerative changes occurred in the tissues prior to the deposit of the urate compound. While some observers maintain that such degeneration is a primary factor (Ebstein and his school), others hold that urate of soda can be deposited in unchanged living tissues (Riehl, Lukhatschiff, and His). The subject is a complicated one, and requires further elucidation. While the uratic deposits are seen most characteristically in joint structures they are by no means confined to them. Nearly all the structures of the body which contain a large amount of connective tissue have been found affected. In the central nervous system, crystals of urate of soda have been found in the dura and pia mater, in the neurilemma of nerve-sheaths, in patches of cerebral softening, and in the cerebro-spinal fluid. A similar condition has been noted in the cardio-vascular system, the aortic and mitral valves and aorta occasionally showing a deposit. Among other sites may be mentioned the eyelids, sclerotic, auricle, tendons and tendon sheaths, vocal cords, bronchi, bursæ, bone marrow, palmar, and other fascia, and subcutaneous tissue generally. Indeed it is probable that a careful examination of the various organs and tissues in pronounced cases of gout would reveal the presence of a deposit in many other places not previously described. These points sufficiently indicate that uratic precipitation is very variable in its incidence, a fact of considerable import in connection with the phenomena of irregular gout. Before considering the changes in articular structures a few words may be said about the deposition of urate of soda.

The presence of urate of soda may be manifest as a subcutaneous infiltration of the connective tissue and tendon sheaths (*e.g.* in Dupuytren's contraction), or it may be present in the form of localised subcutaneous swellings—tophi. In yet other cases it may be a component part of an enlarged, thickened, and inflamed bursa, in which case the swelling may reach a considerable size. *Tophi* vary in size from a very minute deposit, the size of a pin's head, up to or exceeding that of a small orange. Among the more common situations are the ears, fingers, foot, ankle, and eyelids, but they may be seen in other situations. The general appearances vary according to the site and the presence or absence of ulceration. Their consistency and chemical composition are also variable. The term chalky deposits usually applied, while giving a fair general impression of their appearance, is not strictly accurate, as there is no carbonate of lime in their composition.

Comparatively few detailed observations have been made on their chemical composition. The results obtained by Langin, Wurzer, and Lehman are given in the following table:—

	Langin.	Wurzer.	Lehman.
Sodium urate	25·92	29·70	52·12
Calcium urate	15·75	29·30	1·25
Sodium chloride	16·70	18·00	9·80
Potassium chloride	...	2·20	...
Calcium phosphate	...	...	4·32
Animal matter	16·70	19·50	28·49
Water / Undetermined residues	19·60	10·30	3·88

Lehman's specimen was supposed to include a fragment of bony substance. Calcium oxalate has also been found in large amount. The salts of uric acid, which are the important ingredients, can be readily recognised by the following reactions:—Take a fragment of the tophus and dissolve by the addition of a few drops of nitric acid. Evaporate slowly almost to dryness in a porcelain capsule, when, if uric acid be present, a yellowish deposit appears, and on addition of one or two drops of ammonia a rich purple

colour is exhibited — purpurate of ammonia. This constitutes the well-known murexide reaction.

When present in the subcutaneous tissue in a diffused form the uratic deposit may not be readily observable to the naked eye, but its presence may be suspected from the associated thickening and contraction of the tissues, and by coincident ulceration. A correct diagnosis may only be determined by a microscopic or chemical examination, the former revealing characteristic needle-shaped crystals of urate of soda, the latter being readily determined by the murexide test.

The Joints.—When a gouty joint is examined in the earlier stages of the disease the articular cartilages show scattered or isolated points, streaks or patches of a "chalk-like" material—urate of soda. A closer examination may reveal the fact that this deposit is not really on the surface of the cartilage, but is situated interstitially in its substance, the superficial layer of epithelium being intact. Later this superficial layer is involved, and the articular surface becomes roughened, irregular, and eroded. As the primary deposits take place in the areas where circulation and nutrition are at their lowest level, we accordingly find that it is the central portion of the articular cartilage that shows the earliest manifestations. As the disease progresses the ligamentous structures become the seat of interstitial deposit, and in inveterate cases they may become extensively destroyed. Before this has taken place, however, the synovial membranes and their fringe-like processes are involved, the surface of the synovial membrane appearing as if dusted over with powdered plaster of Paris. The rest of the synovial membrane is usually congested, thickened, and thrown into irregular folds. The synovial fluid may be in excessive amount, and may be turbid from the presence of specks of urate. An acid reaction has been observed in one instance. In very rare instances the contents of the joints have been found hæmorrhagic or purulent. The ends of the bones frequently become enlarged, and true bony ankylosis occasionally occurs. Changes also take place outside the joints, the connective tissues, aponeurosis, and tendon sheaths becoming the seat of a varying amount of uratic infiltration. The bursæ in the neighbourhood of a joint may be extensively involved independently of any joint affection. Secondary changes are induced in the joint structures by its occurrence. Inflammatory changes occur in the cartilage leading to proliferation and necrosis, the proliferative changes being most manifest along the borders of the cartilage where the deposits are smaller and the tissues more highly vascularised. As a result we have outgrowths at the margin of the articulation, best seen in the larger joints. Some degree of osteitis also occurs with degenerative changes in the bone and marrow, and a varying degree of uratic infiltration. Wynne, quoted by Duckworth, holds that the hypertrophic outgrowths (lipping) present in gout must be regarded as true exostosis, and not ecchondroses as in rheumatoid arthritis.

The microscopic appearances of the cartilages are fully described by Duckworth, who is of opinion that there is no microscopic appearance of cartilage characteristic of gout.

Before leaving the consideration of the joint changes attention may be drawn to an important contribution by Moore on the subject of uratic deposits in joints. Moore (*St. Bartholomew's Hospital Reports*, 1888) made an extended series of observations into the condition of the joints, small and large, in over seventy post-mortems. In some of the cases recorded no gouty history had been obtained during life. Two of his conclusions have a very special bearing on the points under consideration :—

(*a*) It is common to find urate of soda in the joints of those persons whose aortic valves show chronic degenerative changes with calcification, and who therefore belong to the class of patients likely to have had angina pectoris.

(*b*) Urate of soda is present in the joints of a large proportion of those persons over forty years of age who die of cerebral hæmorrhage.

The importance of these observations is not perhaps sufficiently recognised in this country, where they serve to justify a diagnosis of gout in not a few cases where joint changes are entirely latent. But they are of still greater importance from the point of view of the opinions held by various foreign writers on the too frequent diagnosis of gout by British physicians. While admitting that a diagnosis of gout is frequently arrived at on insufficient grounds, these observations prove that it is not advisable always to withhold a diagnosis of gout (and substitute rheumatism) except in cases where there has been a previous acute attack, or where there is distinct clinical evidence of uratic deposits.

Visceral Changes in Gout.—The only visceral disease which is intimately related to gout is contracted granular kidney, and the relationship is a complicated one. In some cases the renal disease precedes the articular manifestations; in others it seems to appear as a sequel, and in a few the conditions appear and develop simultaneously. Moore found that chronic interstitial nephritis was not invariably accompanied by deposits in the articular cartilages. Ord and Greenfield found that in a series of cases of gouty affection of the great toe joint, in two-thirds of them there was a definite co-existence of contracted granular kidney, and in the remaining third there was an affection of the kidney closely allied to it. The question is a difficult one, the difficulty being partly due to the fact that whereas a diagnosis of chronic

renal disease is frequently arrived at without much difficulty, in other cases this is not so, and it may be impossible to affirm that the kidneys are not diseased. Gouty kidneys may show a few marked deposits of urate of soda both in the cortex and medulla, the deposit being situated in the intertubular connective tissue, and only occasionally in the tubules.

Typically gouty deposits are also occasionally met with in the heart (once), arteries, and veins, and in other situations; but as these are rare, and the associated cardio-vascular diseases are very common, no great stress can be laid on their occurrence. In the course of the disease numerous other morbid changes affecting different structures and organs are met with, but as they are referred to under chronic gout they do not call for detailed reference.

Gout in Lower Animals.—It is obvious that if positive results could be obtained in the production of gout in lower animals, very valuable information might be gained from them regarding the etiology and treatment of the disease. So far, however, observations on these lines have been too limited to be of much practical value.

The investigations of Zalesky and others, which were originally undertaken about the year 1850 with the view of elucidating the part played by the kidney in the formation of uric acid, gave results which agreed in demonstrating the presence of deposits of uric acid salts in various organs and tissues.

In 1882 Ebstein carried out an elaborate series of experiments, the results of which led him to formulate the theory associated with his name, viz. that necrotic changes in the tissues are the primary cause of gout, the necrosis being due to the presence of dissolved urates in the fluids of the body. One set of experiments consisted in the ligature of the ureters of cocks; a second series comprised the subcutaneous injections of small doses of neutral potassium chromate. By the first means he prevented the elimination of the urates, and so led to their being dammed back and retained in the tissues; the potassium chromate was regarded by him as exercising a damaging influence on the renal epithelium, in virtue of which its excretory activity was impaired, with the result that a similar although less acute retention of urates occurred. The fowls died within twenty-four hours after the ligature of the ureters, but lived for a few weeks after chromate administration. Post-mortem examinations in both instances revealed the presence of uratic deposits in the articulations, tendon sheaths, liver, muscular tissues, and serous membranes, these changes being much more pronounced in the chromate experiments. Ebstein, however, considered that the differences were those of degree only, and corresponded to the different lengths of time the animals survived. It has been urged against these experiments that, as uratic deposits were prominent in the liver, muscular tissues, and serous membranes, localities which are not similarly affected in human gout, the two processes cannot be regarded as wholly comparable. While the possible validity of this objection must be recognised, we must also admit the possibility that the conditions found by Ebstein are really analogous to human gout, the difference being only one of degree.

In 1888 Mendelson published an interesting paper on guanin gout in the hog and its relations to the sodium urate gout of man. This subject had been primarily referred to by Virchow and Roloff, but the observations of these observers were not elaborated. Mendelson found numerous small discrete, chalky-looking masses of guanin in the periosteum of bones, and on the surface of the junction between the epiphyses and the shafts. Similar deposits were abundant in the ligaments and peri-articular tissues generally, and also scattered indiscriminately in the cartilage covering the ends of the bones and semilunar cartilages. The muscles showed like deposits, especially in the intermuscular septa. In no instance were any signs of inflammatory reaction about the site of deposit discovered, which circumstance, considered along with the absence of structural change in the tissues immediately around the areas of crystallisation, led Mendelson to conclude that there was a very gradual accumulation of the deposit, allowing the tissues to adapt themselves to its presence. This observation led him to the belief, in opposition to Ebstein's view, that the deposit was primary and the necrosis a secondary development. This guanin gout is an exceedingly rare condition. It may be identified as follows:—Evaporated upon platinum foil with a drop or two of strong nitric acid, a slimy, yellowish red residue remains, which, if touched when cold with a drop of sodium or ammonium hydroxide solution, becomes of a deep reddish brown colour, changing to dark purple on heating (Hoppe-Seyler). This test is quite distinct from the murexide reaction of uric acid. I am informed by large importers that American hams sometimes show the presence of minute specks suggestive of the description given by Mendelson, but so far I have not been able to discover any specimens.

The latest recorded experiments by Kossa and Kionka have been more dietetic in nature. Kossa in 1899 fed birds on cane sugar and dextrose, and succeeded in inducing so-called avian gout. This consisted in the appearance of crystals of sodium urate in the looped tubules of the kidney, followed sooner or later by renal inflammation. There also developed in the tissues a condition similar to that described by Ebstein in his chromate experiments. He also found a marked increase in the nitrogenous metabolic products, and a point of some interest therapeutically was determined by him, viz. that

the animals so affected died all the sooner if they were treated with piperazin.

The administration of oxalic acid and its salts, corrosive sublimate, acetone, and other substances, also induced these appearances of avian gout, although in a milder form. With regard to dogs and rabbits, it was observed that the subcutaneous injection of sugar did not produce a similar result.

In 1898 Minkowski, by feeding dogs with adenin, a substance closely allied to uric acid, induced a deposit of urate of soda in the kidneys. His observation is a suggestive one, and a confirmation and extension of this line of inquiry is desirable. At present, however, the difficulties in the way of procuring adenin are almost insuperable.

The most recent and probably the most interesting experiments yet recorded are those of Kionka (1900). After a preliminary reference to the occurrence of avian gout in fowls, ostriches, and birds of prey, he goes on to describe his own experiments. These consisted in feeding full-grown fowls on a diet of minced horse flesh (freed of fat and gristle) and water. He found that after a period of three to five months a disease developed which gradually assumed the characters of real gout. Several types of disease were observed. Some fowls exhibited a more rapid form of the disease, characterised by weakness of the lower limbs, loss of appetite, unsteady gait, and swelling of the joints, these symptoms being aggravated at intervals in a manner suggesting acute attacks. Death occurred eventually from exhaustion. On post-mortem examination the joints were swollen, œdematous, and showed deposits of urates. These deposits were found to be more pronounced in the more chronic cases, in which the attacks had not been so manifest, and where the most marked features were the development of tophi in the joints and webs of the feet. In other cases the features of visceral gout were manifested, uratic deposits being present in the serous membranes of the intestine, and uric acid infarcts in the kidney. In all the cases the usual appearances post-mortem were those of gouty kidney.

From some points of view it is unfortunate that a more detailed account of the histological characters of the organs and tissues affected, and a fuller record of the end products of metabolism, are not given in this interesting paper. This line of inquiry is one of great interest, and as it is important that the observations be repeated and extended, I have arranged to do so, and am at present engaged with it. At the present time, after nearly two months' treatment, the fowls under observation are not obviously gouty, although there are indications that some of them are not in perfect health. The results of the investigation will be described in the article "Uric Acid."

Gout is usually described, although in rather vague terms, as one of the rarer diseases of a poultry yard. I have taken some pains to obtain information from the leading poultry experts in this country, but cannot get unequivocal evidence in reply. One expert writes: "A swollen condition of the feet, attended with heat, to which fowls are liable, has been wrongly described as gout. Such a condition is simply due to congestion of the blood-vessels consequent on a plethoric state of the system, and birds in confined runs, when exercise is limited, or practically prevented, are very susceptible to stagnant circulation in the limbs and feet, which produces these supposed gouty symptoms." On the other hand, another writes:—

"This (*i.e.* gout) is rather liable to be mistaken for leg weakness, but may be distinguished by the legs and feet feeling hot, with evident swelling, and a more or less inflamed appearance. It is chiefly found in Asiatic breeds. The bird should be removed to a dry warm place, and given a dose of jalap or calomel to open the bowels, after which a half-grain pill of extract of colchicum should be administered twice a day. The legs and joints may be well rubbed with sweet oil daily with benefit." Other references bear out this latter statement. I make no apology for entering so fully into this matter, as it is one of practical importance, especially as the reader may have opportunities of testing the accuracy of these statements, and so add to our knowledge of the subject.

Acute Gout.—The manner of onset, course, and subsequent history of acute gout varies within such wide limits that no description, however detailed, can be made applicable to each individual case. Peculiarity of constitution, whether hereditary or acquired, continually varies the aspect of the disease, the natural course of which is doubtless also undergoing invisible yet none the less important modification under the influence of improved hygiene, greater muscular activity, and the slightly improved habits of eating and drinking of the present day compared with those obtaining in the past.

With regard to a description of the acute attack I cannot do better than give Sydenham's graphic description, based as it is on direct personal knowledge of the disease as well as on extensive clinical experience otherwise. This description is minute and accurate even at the present day, it is historically of great interest, and the little admixture of theory shown in the reference to digestion and dispersion of the peccant matter does not deduct from the value of his clear and interesting record:—

"Suddenly and with scarcely any premonitory feelings the disease breaks out. Its only forerunner is indigestion and crudity of the stomach, which troubles the patient for some weeks previous to the acute attack. His body also feels swollen, heavy and windy, symptoms which

increase from day to day until the fit breaks out. A few days before this torpor comes on, and a feeling of flatus along the legs and thighs. Besides this there is a spasmodic affection, whilst the day before the fit the appetite is unnaturally hearty. The victim goes to bed in good health and sleeps. About two o'clock in the morning he is awakened by severe pain, generally in the great toe; more rarely in the heel, ankle, or instep. This pain is like that of a dislocation of the bones of those parts, and is accompanied by a sensation as of chilly water poured over the veins of the suffering joint. Then follow chills and shivers and a little fever. The pain, which was at first moderate, becomes gradually more intense, and while it increases the chills and shivers die out. Every hour that passes finds it greater, until at length at night time it reaches its worst intensity, and insinuates itself with most exquisite cruelty among the numerous small bones of the tarsus and metatarsus, in the ligaments of which it is lurking. Now it is a violent stretching and tearing of the ligaments, now it is a gnawing pain, and now a pressure and tightening. So exquisite and lively meanwhile is the feeling of the part affected that it cannot bear the weight of the bedclothes nor the jar of a person walking in the room. Hence the day is passed in torture, and a restless rolling, first to one side then to the other, of the suffering limb, with perpetual change in posture; the tossing of the body being about as incessant as the pain of the tortured joint, and being at its worst as the fit is coming on. Hence the vain efforts, by change of posture both in the body and the limb affected, to obtain an abatement of the pain. This goes on towards the second or third hour of the morning (a whole day and night after the first outbreak of the fit), such time being necessary for the moderate digestion and dispersion of the peccant matter. The patient thus has a sudden respite which he falsely attributes to the last change of position. A gentle perspiration is succeeded by sleep.

"He wakes freer from pain, and finds the part recently affected swollen. Up to this time the only visible swelling had been that of the veins of the affected joint. Next day (perhaps for the next two or three days), if the generation of the gouty matter has been abundant, the part affected is painful, getting worse towards evening and better towards morning. A few days later the other foot swells and suffers the same pain. The pain in the latter regulates the state of the one first attacked, for the more acutely it is tortured, the more perfect is the abatement of suffering and the return of strength in the other. Nevertheless there is a repetition in the second case of all the misery of the first, both as regards intensity and duration. Sometimes during the first days of the disease the peccant matter is so exuberant that one foot is insufficient for its discharge. It then attacks both and that with equal violence. Generally, however, it takes the feet in succession."

With regard to *premonitions* there is a great diversity in the nature and frequency of their occurrence, and in a few cases none may have been observed. Those most frequently encountered have reference to the digestive system, the usual run of symptoms in a robust adult being as follows:—

The patient does not feel well, but cannot clearly define his feelings. He feels "gouty" and thinks you should understand the feeling. On closer inquiry we find that there has been some slight irregularity in the intestinal action, a little undue peristaltic movement associated with slight discomfort not amounting to pain, slight constipation, a moderate degree of flatulence, with fitful sleep the preceding night. The tongue is slightly furred posteriorly with a faintly yellowish white fur, the breath is a little foul, the appetite unimpaired or capricious, the conjunctivæ a little muddy, and the pulse tension slightly raised. If he has had many previous attacks, there are other subjective sensations in the arms or legs. His facial expression reflects the coming trouble, and, as the mind is preoccupied with the analysis of these various disturbances, some depression of spirits or irritability of temper is a not unnatural sequel. In other cases various nervous disorders are the most prominent indications—shooting pains, cramp, neuralgia, headache, cardiac irregularity, and irritable temper.

The *constitutional disturbance* is as a rule out of proportion to the amount of febrile movement. The temperature does not usually rise above 102°, but varies with the severity of the local inflammation, and to a less extent with the number of joints involved. The fever remains from three to five days, but may remit and recur. The acute attack may occur at any period of the day, and while the usual course of the disease is for the great toe joint to be the part first affected, in not a few cases the primary attack occurs in the knees, ankles, tarsus, or hands. This fact may be explained in some cases by the previous occurrence of an injury to the part affected, in virtue of which its vitality has been permanently weakened, and the part rendered more prone to the incidence of the existing poisons. In other cases, however, there is nothing discernible either in the previous history or occupation of the patient to explain this departure from the usual course of the disease. When the swelling increases the pain lessens; later the parts pit on pressure, and the cuticle cracks and desquamates. At the height of the attack the pulse tension is usually high, the tongue heavily coated, the appetite gone, hiccough, eructations, sometimes vomiting, and also constipation present. In very severe cases, where the affection has ceased to be a purely articular one, I have observed

that pain in the interior of the bones was a distinct feature. Only very exceptionally does suppuration attend the gouty inflammation. The state of the blood and urine is referred to later (p. 499).

While acute gout is commonly a disease of adult life, not appearing as a rule under the age of thirty-five, there are numerous exceptions to this. In cases where acute manifestations of the disease develop at an earlier date, from the late teens onwards, the paroxysm is less declared; not infrequently all that is manifest is a slight attack of monarticular pain, with redness, swelling, and distended veins, lasting for a varying number of hours. Such cases may develop and recur without ever culminating in the classical picture of an acute paroxysm. In severe cases of confirmed and recurring gouty manifestations of an acute nature, metastasis is of very frequent occurrence.

The *subsequent history* of an attack varies within wide limits, and is mainly dependent on the hereditary quality of tissue in each individual. The more speedily the constitutional disturbance is concluded the less is the risk incurred of the gouty diathesis becoming confirmed, and the greater the security against the associated ailments entailed by protracted or oft recurrent gout. If appropriate treatment be not assiduously carried out the disease tends to recur, and acquiring force by repetition eventually inflicts its wonted penalties. Attacks may recur every year, or even much more frequently, and indeed there is no limit to the irregularities in the frequency or severity of their occurrence. As a favourable illustration Sir W. Roberts described a case where the first attack, a typical one, occurred at the age of twenty-nine, and the next one in the patient's eighty-ninth year.

The *subsequent local changes* similarly vary. Sir Dyce Duckworth has described a case in which careful examination of the joint previously affected revealed no evidence of gouty deposit. On the other hand, we know from Moore's observations that gouty deposits frequently exist in joints that have not been recognised clinically to have been affected with gout.

In very severe and chronic cases the weakness in a limb, recovering from an acute attack, is very considerable, and may only be partially recovered from after several months. Such cases are usually accompanied by muscular wasting, and possibly also by nerve degeneration, and are usually seen in patients whose natural vigour, originally weak, has been much reduced by the ravages of the disease.

Chronic and Irregular Gout

It is probably inadvisable to attempt to differentiate these two conditions too closely. To illustrate. One man has a first attack of genuine articular gout when æt. 20, and in five years or so he is almost a cripple from the disease. Another man may have the first acute manifestations when æt. 28 or 30, and have ten or twelve typical acute attacks before the age of 40, and yet at that age there may be no external evidence of articular or other abnormality. A third man æt. 40 may have suffered from well-marked symptoms of irregular gout for ten or fifteen years, but has never experienced an acute attack, and, like the preceding case, may show no distinct external evidence of the disease. The difference between these three clinical types is one of degree and not of kind. If we selected at random a dozen fairly illustrative cases of the last-mentioned group, and experimented with them as with the lower animals, with the object of inducing typical acute attacks, we would probably be successful in most if not in all the cases. The degree of success would be proportionate to our skill in attaining, without *other unsought constitutional disturbance*, the environment in the digestive tract and in general cell metabolism appropriate to the development of the disease, and the failures would be explained either by faulty methods of experiment, or, occasionally by special idiosyncrasies of the patient. The experiment would doubtless be of further interest as showing different modifications of the acute phases of the disease, corresponding more or less to the great variety in the irregular manifestations originally present.

Just look for a moment at a few of the more important factors which modify the appearances of chronic and irregular gout. These are—

(i.) The force of heredity, and the purity of the gouty strain. The last is a factor of very far-reaching importance.

(ii.) The state of the digestive tract, *e.g.* is it tolerably good, as it often is in cases of pure gout, or is it naturally more or less seriously weakened by hereditary weakness (gouty or otherwise)? The latter group furnishes a familiar class of cases, usually thin, more or less dyspeptic-looking individuals with symptoms mainly gastro-intestinal or nervous. The weak digestion seems to act in these cases as a natural safeguard against the more acute articular manifestations.

(iii.) The state of the nervous system is an all-important factor in the disease, and especially in its irregular development. A recent clever novelist, in referring to the early demise of one of her feminine characters, recorded the death as due to the combined effects of ennui and luxury. Neither alone, the authoress states, would have been sufficient to kill her, but she could not stand against the allied forces. A fairly accurate lay-picture of not a few cases of irregular gout.

The severity of the symptoms is by no means proportionate to the extent of the gouty deposits. This is well illustrated in case 4, p. 519, where

the deposits were very pronounced, and yet gave trouble mainly through their bulk. In other cases the deposits are scanty and the symptoms are severe. It has been suggested that these varieties depend on the fact that the amount of disturbance induced depends more on the site than on the extent of a deposit.

Keeping these different factors in mind, it is obviously impossible to present anything like a complete picture of the chronic and irregular phases of the disease, and it must suffice to give merely a short outline of chronic gout, including the deforming and tophaceous variety with the associated cachexia, and thereafter an equally brief outline of the phenomena of irregular gout as seen in the various systems of the body.

Chronic Deforming Gout.—Here the articular changes are very pronounced, and are usually most marked in the joints of the fingers, ankles, knees, and great toe, but they are by no means confined to these structures. There is also found uratic infiltration with degeneration in various fasciæ, bursæ, and tendons in the neighbourhood of the affected joints, and indeed in not a few cases these sites are the primary ones. In a few instances the greatest deposit may be in one or other of the important bursæ in the region of a joint, and such cases may be less amenable to treatment than those in which there is a more diffuse articular change. The deformities induced are those due to the swelling of the joints, deflection of the fingers, hands, and toes, ankylosis and other local changes, and they closely simulate the appearances in rheumatoid arthritis. (*See* "X-Rays.") *Nodi digitorum*, the term applied to the knotty or knobby state of the terminal phalangeal joints by Heberden (Heberden's nodes), are frequently of a gouty nature.

Chronic Tophaceous Gout is characterised by deposits in the subcutaneous tissues, notably of the ears, in the neighbourhood of joints, and in various bursæ. These tophi may give way and exude a characteristic creamy fluid. They may be present in large size in the neighbourhood of a joint, which otherwise appears free from the disease and whose mobility is unimpaired.

Gouty cachexia is the sequel of chronic gout in certain predisposed subjects, but the causes of that predisposition are not quite clear. It is seldom met with in the robust, plethoric subjects who present the classical pictures of the disease, and is more usually seen in cases where the disease develops early in subjects strongly predisposed to it. The symptoms of the cachexia are numerous and varied, but in the main are those of cachexia from any other cause. The anæmia and associated degenerative changes in the muscular and nervous tissues in turn react unfavourably on the gouty tendency. The functions of all the muscular, glandular, and nervous organs are profoundly deteriorated, and the results in any given case vary according to the extent of the degenerative changes in one or other or all of these organs. The prognosis is bad when the cachexia is doubtful.

Irregular Gout—Goutiness

The symptoms of irregular gout are manifold and varied, and are not infrequently most developed in persons who have never been the subject of the acute phases of the disease, and in those who have no pronounced evidence of chronic gout. Are we justified in the diagnosis in these cases? Certainly. While we must avoid ascribing all diseases occurring in subjects with a hereditary history to a gouty origin, a careful study of the heredity, constitution, and clinical features in many cases will amply warrant the diagnosis. The views entertained by not a few writers that a diagnosis of gout is not possible in the absence of tophi or a history of previous acute attack are not to my mind tenable. But a careful discrimination must be exercised in this direction.

To what are the symptoms of irregular gout due? Can they all be explained by the view that they are dependent on the local deposit of minute crystals in the affected parts, *e.g.* gastric mucous membrane, cerebral membranes? Or are they dependent in whole or in part on the selective action of a soluble toxic substance in the blood on a naturally weak spot? A final answer cannot yet be made. The symptoms vary with the age and constitution of each subject, but in every case we may take it that the symptoms are more liable to develop in that system or tissue which happens to have, from natural or acquired defects, the weakest nutritional activity. Here again we must have regard to the influence of other hereditary strains in modifying the direction and severity of the gouty symptoms.

The Nervous System

We are only beginning to realise how often gout acts on many organs and tissues, and how frequently inflammation is thus induced (Gowers and Taylor, 1899). This opinion well illustrates the importance of the association between gout and diseases of the nervous system, and at the same time it indicates the limited extent of our precise knowledge of the subject. The effects of gout are seen both on the higher and lower neurones. *Neuritis* is by no means infrequent, and is probably most encountered in the third and fourth decades of life, but it is probable that many cases of supposed neuralgia in young subjects are really minor forms of neuritis. Sir Willoughby Wade was of opinion that a neuritis of the nerves at the affected part was a feature of the acute attack, but I have never found the distinct linear tenderness described by him. Some time ago, while experimenting with electrical currents on the hand of a gouty friend, I was much interested to find that the cutaneous sensibility in a finger whose joints were the seat

of articular changes was much diminished. The point is worthy of further investigation.

Myelitis is rare. Gowers considers that possibly some cases of otherwise undetermined origin in young adults owe their origin to this cause. In *neuralgia* we find the most common, as it is also one of the most troublesome, nerve derangements. Its relationship to gout, and its association with other traits of the disease, are frequently perfectly apparent and unmistakable, and would probably be more so if sufficiently detailed inquiries were instituted. The fifth nerve, posterior tibial, sciatic, and occipital are the ones most frequently involved, and their derangements are liable to appear and disappear suddenly, or take the place of other manifestations of the disease. Cases like the following are instructive and by no means rare:—A young man æt. 32, of plethoric build and hair grey almost to whiteness, came complaining of "acidity." His father had been a martyr to gout and in his later years was affected with attacks of angina pectoris. When 25 years old this patient had a very severe attack of neuralgia which fully prostrated him for two or three weeks, and incapacitated him from work for as many months, and at the end of this time his hair was "almost white." Lesser attacks have occurred subsequently, and, curiously enough, the prodromal symptoms were the same as those experienced by his father—a feeling as if the right thigh was being sponged over with cold water. From the great severity and long duration of the first attack it may probably be regarded as a regular acute gout in an unusual site. There is indeed no end to the irregularity and anomalous nature of the nervous disturbances encountered. In young adults *tender feet* may be an early symptom, and is usually accompanied by the hot sensations in the feet described by Duckworth. The discomfort arising from this condition in young women may be so great that they will never wear boots or outdoor shoes a minute longer than they can possibly help. In one such case I have observed a localised point of exquisite tenderness over the middle of one calf, and this patient averred that the feelings of *cramp* to which she was subject seemed to have their origin at this point. This case was specially interesting, as the patient was by no means of a neurotic temperament.

The *local phenomena of Raynaud's disease* are also frequently seen in the subjects of inherited gout, the pallor and coldness of the affected extremity persisting from a few minutes to half an hour or more. These patients are usually very susceptible to the influence of cold water, and cannot tolerate sea-bathing. In adult life *vaso-motor instability* may be a prominent feature, especially in plethoric subjects.

Headache, *migraine*, *insomnia*, and *vertigo* are also frequently encountered as the leading subjective indications of gouty metabolism, and they may be accompanied by feelings of physical and mental lassitude, or melancholia. All these cases may be associated with some obvious aberrations in urinary excretions. At and after the climacteric we sometimes find that patients who have been treated for years for recurring acute and subacute gout of a typical character, recover completely from the symptoms, but develop more acute *mental derangements,* necessitating appropriate and permanent asylum treatment. Are these cases to be regarded as illustrations of metastasis, or as illustrating a reversion to another type of inherited weakness under the influence of the diminishing metabolic activity of later life?

The liability of many gouty subjects to *cerebral hæmorrhage* need only be referred to, and it may be well to mention that in old-standing cases of hemiplegia the deposit of urate of soda takes place more readily in the joints and tissues of the paralysed side.

Circulatory System

In early life the subjects of inherited gouty proclivities frequently exhibit various vascular disturbances, *e.g.* chilblains, and the local phenomena of Raynaud's disease. In later life the leading manifestations are the following:—

(i.) General vaso-motor instability.

(ii.) Incidental and more or less frequent aberrations in cardiac action, arising from the influence of the toxic agents on the nervous mechanism of the heart, *e.g.* arrhythmia, tachycardia, bradycardia, angina. The tendency to these disturbances is, of course, increased if there be myocardial degeneration.

(iii.) The symptoms and physical signs arising from the action of a hypertrophied heart, with or without dilatation, and contraction of the peripheral vessels, in cases with renal cirrhosis and widespread arterial disease.

(iv.) Evidence of early or late cardiac muscle failure, due to chronic myocarditis, with disease of the coronary vessels.

These various symptoms are frequently induced or aggravated by reflex injurious influences from the gastro-intestinal tract, and in many of these cases the dietetic treatment is the all-important one. This is specially true of the senile heart. Pericarditis is by no means an uncommon manner of termination in cases where the gouty form of Bright's disease is developed. In addition, various arterial and venous disorders are met with. Arterio-capillary fibrosis, and atheroma and their sequelæ, are the arterial derangements; phlebitis and thrombosis are the leading venous disorders. Varicose veins in the legs and hæmorrhoids are by no means infrequent. While the diagnosis of a gouty origin in many of these cases is often largely a matter of inference from the *tout ensemble* of the case, the fact of the occasional relationship of these symptoms to gout should not be lost sight of.

Alimentary System

The gastro-intestinal tract is in the closest sympathy with the cutaneous system, and there is every reason to believe that the various nervous and vascular disturbances in the latter are represented by analogous changes in the internal continuation of the epithelial covering. There is no doubt that some of the most troublesome cases of irregular gout are those whose symptoms are referred to the digestive tract, and nowhere more than in this system do we find good illustrations of the doctrine of metastasis. The nature of the symptoms varies considerably in different cases. In one case they are sudden in onset, sharp and fugitive in nature, and give place to definite articular or other manifestations. For example, a sudden *gastralgia* with associated derangement of the digestive functions develops, persists, it may be, for a few days, and suddenly disappears, coincidently with the development of an eczema, an arthritic attack, or an urticaria, and *vice versa*. Acute attacks of abdominal pains (*enteralgia*), with a varying amount of constitutional disturbance, are also seen in undoubtedly gouty subjects, and the clinical appearances and course under treatment may leave no room for doubt as to the accuracy of the diagnosis.

Apart from these more purely nervous manifestations, catarrhal states of the mucous membrane are prone to occur. *Pharyngitis*, acute or chronic, is the most common, but gastro-enteritis is also met with. The very acute congestion and swelling of the pillars of the fauces, uvula, and tonsils, often with distension of the superficial veins, form a quite characteristic picture. Parotitis is a rare incident in the gouty, and when present seriously interferes with mastication and deglutition. It usually develops suddenly, persists for a few hours, and disappears.

In the more asthenic type of the disease the *dyspeptic symptoms* are more those of chronic enfeeblement of the digestive powers, the more important signs being epigastric pain or discomfort, attended by waterbrash and slight flatulence ("acidity"), some depression in spirits, and other evidences of deranged metabolism. This class of case is specially liable to show some obvious urinary abnormality, *e.g.* lithæmia or phosphaturia, which conditions may alternate.

In other cases we find slight attacks of gastric catarrh, usually termed biliousness, characterised by loss of appetite, furred tongue, foul breath, and constipation.

While we must admit that all these symptoms are commonly seen independently of gout, this should in no way interfere with our recognition of their gouty origin in many cases. A few authentic cases have been recorded which revealed unmistakable post-mortem evidences of uratic deposit in the alimentary tract, and such are probably present, though in very small amount, to a greater extent than has been described.

And further, we must bear in mind that the uratic deposit is a late sequel and an inconstant feature of the disease. Its presence is conclusive proof of its existence, but its absence in no way negatives it. We must remember the incessant nature and also the rapidity of the chemical changes occurring in the digestive tract, the liver, and tissues generally. If the views put forward under etiology are correct (p. 501), the relationship between various foods, liquids, etc., and gouty symptoms becomes a little more clearly defined and understood.

A careful study of the general and hereditary history and clinical features of each case will usually suffice to determine its exact nature, and the more thoroughly various minor disorders are studied, the more evidence will they frequently afford of their origin from a common stock.

To what extent are the so-called functional diseases of the liver associated with gout? Alike from the important action of the liver on the carbohydrates of the food, and in the formation of urea (*vide* "Liver, Physiology of"), we have ample evidence in support of the view that in gout, and perhaps even more particularly in irregular gout, liver derangement plays an important part. But the exact nature of the derangements in function induced by temporary vascular and other alterations is quite unknown. It is complicated and probably also inconstant, and with our present knowledge, we must be satisfied with a few general reflections gained from empirical treatment (*vide* "Liver, Diseases of, Functional"). This will be further referred to under "Uric Acid."

The last point that calls for reference is constipation. Gouty subjects are much more prone to the injurious effects of constipation of even a slight degree than non-gouty individuals.

Renal System

The hereditary influences of gout frequently show their earliest indication in connection with the urinary system. Gibbons, in an interesting paper,[1] has drawn attention to the frequent occurrence of renal colic in infants of gouty stock. This is further emphasised by the following cases lately recorded to the writer by a medical friend, himself a martyr to gout. His personal history is as follows: Acute gout, twice, in ball of right great toe, gouty dyspepsia, dry eczema on skin, grooved and fissured nails, and one joint of right hand deformed and ankylosed. Three of his children have exhibited symptoms. The youngest, when an infant, was severely troubled with disordered micturition, and bladder irritation, with passage of much free uric acid; another was troubled when a boy with hæmaturia due to the passage of large

[1] *Med.-Chir. Transactions*, vol. lxxix. 1896.

quantities of uric acid; and a third has had an attack of acute gout in the heel, and in his youth was affected with erythema nodosum. When considering the subject of uric acid excretion in infancy I may draw attention to the statement usually made in most works on pediatrics as to children passing relatively more uric acid than adults. So far I have not been able to trace this to any definite series of observations, and lately I made an investigation on the excretion in a child of 20 months, and the results (as yet unpublished) indicate that the proportion of uric acid to urea in the infant is not essentially different from that in the adult. The point is one that merits further investigation.

Transient albuminuria is met with in gout, especially during the acute attacks, and the amount and frequency of its occurrence may be regarded as evidence of the degree of renal weakness. Similarly transient glycosuria occurs, and may be regarded as similar evidence of hepatic (and possibly also muscular) insufficiency. If pronounced, it may merge into true diabetes.

Oxaluria is a less frequent occurrence. Phosphaturia is by no means rare, and an apparent excess of phosphates occasionally alternates with the passage of large amounts of free uric acid. But it should be carefully noted that the occurrence of oxaluria or phosphaturia often depends entirely on the state of acidity of the urine. If the acidity of the urine is low (sodium phosphate present in small amount) there is a greater tendency to the deposit of the oxalic acid normally present in the urine, and there is also a preponderance of the neutral, not readily soluble, and basic, easily soluble, earthy phosphates over the acid salts. We are not yet in a position to state authoritatively that phosphaturia is to be regarded as an indication of abnormal disturbance in the phosphorus metabolism in the organism.

Ear

The external ears, and the parts around, are a favourite site for the development of gouty eczema, and the auricle is prone to the deposit of tophi. Beyond these two facts there are few definitely determined as conclusive of the relationship between gout and ear diseases, but there are numerous isolated references bearing on the point. Gout is said to exercise considerable influence in the development of exostosis in the external auditory canal (Pritchard). Hang has described a case where the ear was affected by prodromal pains, with hyperæmia and swelling of the auricle for several days, followed on the third or fourth day by an attack of articular gout. Mirk has recorded chalky deposits in the tympanic membrane, and he is of opinion that some cases with subjective noises are due to gouty deposits in the labyrinth. Baum also testifies to the effect of gout as a cause of ear diseases, especially in producing earache at night and tinnitus aurium without deafness. In the absence of more fully detailed clinical and post-mortem records of the condition of the middle and internal ear, we cannot speak authoritatively on the influence of gout on the structures of the middle and internal ear.

Eye

There is no mistaking the important part played by gout in eye affections, although only in a few cases have definite deposits of urate of soda been recorded in the conjunctiva (Garrod) or elsewhere. Conjunctivitis, episcleritis, sclerotitis, iritis, and irido-cyclitis are the most common manifestations, and the worst feature in these conditions is their great liability to recur. In the gouty form of iritis, hæmorrhage into the anterior chamber is not infrequent (Maitland Ramsay). Gouty subjects are more prone than others to suffer from glaucoma, and Jonathan Hutchinson has recorded cases of hæmorrhagic retinitis and optic neuritis which he regarded as of gouty origin. Apart from these obvious disorders, pain in the eye may be a prominent symptom. This takes the form of a sudden acute pain, sometimes described as linear in nature; in other cases there may be a dull ache with slight tenderness or hot and itchy feelings in the eyeball. In these latter cases the ocular tension should be carefully investigated. The eyelids are also prone to be the site of a fugitive œdema.

Cutaneous System

French writers in particular lay great stress on the special liability of gouty individuals to skin affections, the arthritides, as they call them, but in our own literature we find much less belief in the importance of this relationship. Its great importance is undoubted. It would be strange indeed if this extensive vascular connective tissue covering did not react strongly in many ways to the influence of gout. Every experienced family practitioner knows that this is so. He may have seen at one time a severe attack of abdominal pain disappear with the onset of an eczema, or the development of a troublesome and persistent eczema in a limb already the seat of acute or subacute articular gout; or a gouty family of three, one of whom suffered from headache with lithæmia, a second was troubled with supra-orbital neuralgia and eczema, and a third with erythema and nail disturbances. These and the like convince him, from his own experience, that there is a profound relationship between skin affections and gout. Again, he has known of a lichen developing, and persisting in spite of all kinds of appropriate local treatment for years, possibly undergoing slight modification coincidently with the development of some articular

or other lesion, which for the first time suggested that the two conditions had a common origin. At another time he has been strongly inclined to diagnose scarlet fever in a boy (in the Christmas holidays), but, from his knowledge of the delicate, sensitive skin, both in the patient and other members of the family, also from his knowledge of the heredity, and consideration of the dietetic habits incident to the time of year, taken in conjunction with the itching present, he finally commits himself to a diagnosis of dermatitis, and in a few days there developed the feeling characteristic of exfoliative dermatitis.

These are all actual occurrences, and many other suggestive parallels could be cited. Similarly with *some cases* of pruritus, urticaria, psoriasis, and herpes, and indeed his whole clinical experience, judiciously expressed, clearly establishes for him the importance of the relationship. One reason for the smaller amount of attention given to the subject in this country may arise from the fact that primary cutaneous disorders are more frequently found in subjects who are the victims of less declared gout, and too much stress is laid on the absence of so-called definite gouty symptoms. A careful study of many skin diseases from the point of view of their relationship to a diathetic tendency is a present desideratum, and an essential basis for the inquiry is a full recognition of the significance of the various minor and irregular manifestations of the disease, along with a zealous and critical investigation of the hereditary history of each case.

The view advanced by Roberts that, owing to the tolerance of the skin for uratic deposits, the relationship between gout and eczema is less important than frequently believed, must be received with caution. The uratic deposits are an incidental and often very unimportant symptom of the disease, and it is even conceivable that their presence in considerable amount may be a safeguard against any cutaneous disturbances in the immediate neighbourhood, owing to the development of morbid structural changes in the vascular and nervous cutaneous supply, and the secretory glands at the part. Gouty skin eruptions are seen in their most typical forms in cases of chronic or irregular gout, with few external evidences of the disease, and they are probably very closely related to the circulation in the vessels of the skin of faulty end products of nitrogenous metabolism (probably not uric acid), and to a lesser extent to the attempts of the cutaneous glands to excrete these abnormal substances. The importance of external influences must not, of course, be overlooked. They constitute an important allied force.

Respiratory System.—As definite deposits of urate of soda have been found in the vocal cords, arytenoid cartilages, and in the crico-arytenoid ligaments and joints, and crystals of uric acid have been reported in the sputum, we are justified in concluding that many minor symptoms of respiratory disorders occurring in subjects predisposed to gout are in reality of gouty origin. With regard to *catarrhal states of the larynx* our knowledge is well summed up in the following. Whether or not there be a specialised gouty form of chronic laryngitis there can be no doubt that in certain persons of plethoric habit, limited diet, abstention from alcohol, and the administration of Carlsbad salts, hasten recovery, as pointed out by von Ziemssen (M'Bride).

The part played by the bronchial mucous membrane is even more pronounced. A *simple irritability of the bronchial mucous membrane*, evidenced by a loud spasmodic cough, is occasionally a well-marked feature in the female children of gouty parents. Occasionally an *acute bronchitis* develops coincidently with the abrupt cessation of articular pains, more especially in inveterate cases in whom emphysema and a proportionate degree of heart weakness are present. In other cases the bronchitis may give place to some other manifestation. The relationship of gout and *asthma* is an interesting and intricate one. Asthma occurs by no means infrequently in the offspring of gouty parents, and in such cases its occurrence must be regarded as a typical gouty manifestation (we must, of course, have regard to the possible strong admixture of a neurotic strain in the individual). While some cases are by no means readily amenable to treatment, others furnish the most brilliant illustrations of the influence of a physiological diet and a regulated life on the faulty metabolism. In some cases, with a long-standing asthmatic history, true articular gout may develop in late life; in others a typical gouty attack may alternate with a genuine asthmatic seizure; perhaps in the majority no other well-declared phenomena take place. In this latter instance it would almost appear as if one outlet for the action of the products of the deranged metabolism sufficed, and even gave some protection against others. Lastly, we must refer to the occasional occurrence of *hæmoptysis* in subjects with a gouty tendency, either declared or latent, this being associated with marked emphysema and vascular degeneration, a condition first described by Sir A. Clark. In all such cases care must be taken to eliminate a chronic tuberculous lesion as the cause of the hæmorrhage. While gout and active tuberculous disease are not often associated, we are not, to my mind, justified in thinking that there is any antagonism between them. Everything depends on the predominance of one or other strain. It is interesting to note that M. Baumis considered that a gouty father and a tuberculous mother will beget an asthmatic child. The chart at page 505 shows that the oldest son of a gouty

mother by a tuberculous father was a confirmed asthmatic.

Reproductive System.—The female pelvis is rich in connective tissues, its vascular supply is considerable and subject to periodic variations, and its nervous system is more than usually important and complex. Surely we have here a happy hunting-ground for the manifestations of a disease whose end products are deposited in connective tissues, and in whose life-history nervous and vascular disorders occupy a prominent place. Yet this does not seem to be the case. A reference to the indices of not a few authoritative gynæcological works either reveals no mention whatever of the disease or the barest possible reference to it. One cannot help wondering whether this position is arrived at after a careful study of the possible influences of diathetic conditions on the pelvic viscera, or whether the possible importance of such influence is entirely overlooked. The admitted difficulties in connection with its study hardly warrant the latter position. "If the disorders of the uterus and its appendages were studied more particularly with reference to diathetic conditions I am of opinion that some new chapters in their pathology might be written" (Duckworth). This opinion is well worthy of the careful consideration of those who have the special clinical experience necessary to advance our knowledge of this part of the subject.

Muscular and Connective Tissues. — The palmar fasciæ and less frequently the plantar fasciæ are occasionally the seat of gouty disorders (see Dupuytren's contraction under "Fascia" in this vol.), and Jonathan Hutchinson has drawn attention to a juvenile form of Dupuytren's, in which the induration and contraction are limited to one or more digits, and does not involve the palm. The same author has also drawn attention to the induration in the fibrous structure in the dorsum of the penis as being allied to Dupuytren's contraction, both conditions having a relationship to inherited gout. It may be well to refer here to the occurrence of nocturnal priapism in elderly males affected with irregular gout.

Probably some cases of localised myalgic pains, usually regarded as muscular rheumatism, are in reality of gouty origin. Lumbago is so to be regarded in a proportion of cases, and the aches and pains, of a dull and aching or of a sharp and fugitive character, experienced from time to time in the neighbourhood of joints or other tissues which have been the site of injury at an earlier period, are in all probability of a similar nature. The important part played by the structures in bursæ has already been referred to.

Etiology

Previous to the year 1793 the views entertained as to the etiology of the disease are only of historical interest. In that year Murray Forbes suggested that gout developed through an excess of uric acid depositing itself in the tissues. A few years later (1797) Wollaston demonstrated the presence of uric acid in gouty deposits, and from that time until the present, the existence of a definite and important relationship between gout and uric acid has never been seriously questioned. But as to the exact nature of this relationship we at the present time know little more than the opinion expressed by Murray Forbes more than a hundred years ago.

In 1850 Garrod made his classical contributions to the study of the disease. His most important observation was the discovery of uric acid in the blood, but many of his other observations—clinical, pathological, and therapeutic—yet rank as authoritative even at the present day, and all writers and workers on the subject are under a deep obligation to his standard work. For an adequate appreciation of his work the reader is referred to his treatise on the subject. Here I can only indicate a few points of importance with regard to his views on the etiology of the disease, and more especially those dealing with the acute paroxysm. Recent methods of investigation have lately shown that some of his conclusions require revision and modification. From his researches he formulated the following conclusions regarding the acute attack :—

i. The alkalinity of the blood is lowered.
ii. The amount of uric acid in the blood is greater than in the interval.
iii. The excretion of uric acid falls, this being associated with uric acid retention in the tissues, depending on a diminution of the excretory capacity of the kidneys.

These three conclusions formed the basis of his theory of the causation of the phenomenon of the acute attack.

In 1898 the views as to the diminution of the alkalinity of the blood and to the increased amount of uric acid present in the blood during the acute attack were seriously called in question by Magnus Levy. This observer made an extended series of observations in seventeen cases of gout, using the most modern and reliable methods, and in no case did he find evidence of a diminution in the alkalinity of the blood or of an increase in the amount of uric acid as compared with the interval between .the attacks. Levy's observations on these points appear to be conclusive. With regard to the third point the writer has recorded a series of observations, the results showing an actual increase of uric acid excretion during the attack. Similar observations have been made by Pfeiffer, Levy, Badt, His, and others. Taken collectively, these investigations seem to prove that the three aforementioned conclusions are erroneous, and they therefore indicate that Garrod's views as to the etiology of the acute attack must be

abandoned. If this is so, we must start afresh in search of the cause of the acute paroxysm.

In his later writings Garrod adopted the view that the kidneys are the seat of formation of uric acid in health, an opinion which must obviously very materially influence the views put forward as to the etiology of gout. In this view he has of late years been ably supported by Luff, who has formulated the following conclusions from his original observations on the subject:—

i. Uric acid is not normally present in the blood of man and other mammals, nor in the blood of birds.

ii. Uric acid is normally produced only in the kidneys.

iii. The presence of uric acid in the blood in gout is due to its deficient excretion by the kidneys, and to the subsequent absorption of the non-excreted portions into the blood from these organs.

This view as to the physiology of uric acid is in opposition to those entertained by most physiologists, and in addition the writer has published a series of observations which tend to negative these conclusions. His investigation comprised the examination of very large quantities of the blood of birds, and also the liver, kidneys, and spleen, and the results showed the presence of an appreciable amount of uric acid in the blood and also in these various viscera.

In 1882 Ebstein, as the result of an elaborate series of experiments already briefly referred to, came to the conclusion that the essential element in the gouty process was the development of necrotic areas in the tissues, in which crystalline deposits of urate of soda subsequently took place. He regarded the tissue necrosis as the primary condition, and the uratic deposits as a secondary development. He accepted Garrod's view that the uric acid was the causal agent of the necrotic process, his opinion being that the tissues were led to undergo necrotic changes by contact with uric acid *in a soluble form*. The increase in the uric acid was regarded by him as arising in the majority of cases of primary articular gout through excessive formation of uric acid, and he considered that it was only in the rare cases of primary gouty kidney that retention of uric acid occurs. He considered that the excessive formation of uric acid took place in abnormal situations in the body, especially in the muscles and medulla of bones, and he further held that acute or chronic conditions, or even permanent organic changes, may occur if the excessive uric acid in the lymph passages of the skin, bones, or muscles become congested, as it may do in various parts of the body at once. If the congestion in the lymph passages leads to cessation of lymph flow, then there occurred the sudden acute typical attack of gout.

Many objections have been raised to Ebstein's views. Thus the retention of uric acid produced in birds by ligature of the ureters is very much greater than the uric acid retention in man in acute gout. Further, the acid reaction of the necrosing tissues recorded by Ebstein as essential for the deposition of the uric acid salts is not borne out by v. Noorden, who repeatedly found an alkaline reaction of softened tophi. We must also have regard to the opinions of various observers already indicated, to the effect that deposit of urate crystals takes place in *living* tissues, a view diametrically opposed to Ebstein. And lastly, Luff has suggested that, as in Ebstein's experiments, uratic deposits occurred in the muscles, liver, and serous membranes, situations very rarely encountered in the human subject, the two conditions are hardly to be considered analogous.

V. Noorden has advanced the view that the uric acid formation and deposition is a secondary process produced by the presence of a special local active ferment, and quite independent of the amount and the condition of the uric acid found at other areas in the body, and he has renounced the view that the uric acid has a causal relationship to gout.

Haig's views stand alone in their originality and unhesitating boldness (Ewart). He believes that uric acid, or perhaps xanthin bodies, are the root evil of a great many disorders other than those generally accepted as gouty, and he bases his views on observations made on the acidity of the urine and the uric acid-urea ratios taken along with hypothetical conclusions as to the state of the blood and tissue juices. Personally I have never been able to obtain results which harmonise with his records, and he is to my mind unduly credulous in his interpretation of the state of the urine, blood, etc., as ascertained by the methods employed. The beneficial effects of the treatment advocated by him are undoubted in many cases, but the appropriate explanation is not, I think, to be put along the uric acid-urea lines referred to by him.

Sir William Roberts, 1892, as a result of an important series of chemical investigations on uric acid and its various combinations, advanced the view that there are three compounds of uric acid ($H_2\bar{U}$). These are, the neutral urate, $M_2\bar{U}$, where the metal replaces all the displaceable hydrogen; the biurate, $MH\bar{U}$, where half the displaceable hydrogen is replaced by the metal; and the quadriurate, $H_2\bar{U}\ MH\bar{U}$, where one-fourth of the displaceable hydrogen of two molecules is replaced by the metal. The neutral urates do not exist in the body; the biurate is found in the form of biurate of soda in gouty concretions; and the quadriurate he regarded as the sole form in which uric acid exists in normal urine. He inferred that in the normal state uric acid is primarily taken up in the system as a quadriurate, that it circulates

in the blood as such, and that it is finally voided in the urine in that form. He wrote as follows:—In perfect health the elimination of the quadriurate proceeds with sufficient speed and completeness to prevent any undue retention or any accumulation of it in the blood. But in the gouty state this tranquil process is interrupted, either from defective action of the kidneys, or from excessive introduction of urates into the circulation, and the quadriurate, circulating in a medium which is rich in sodium carbonate, gradually takes up an additional atom of base, and is thereby transformed with biurate. The biurate thus produced exists at first in the hydrated or gelatinous modification, but with the lapse of time and increasing accumulation it passes on into the almost anhydrous or crystalline modification, and the precipitation of it becomes imminent or actually takes place (Roberts).

He then made a series of investigations into the conditions which in an artificial parallel accelerate or retard the gradual transformation into the crystalline biurate, and concluded that sodium salts immediately hastened it. As a practical outcome of these observations he strongly deprecated the use of sodium salts, either in the form of medicines or as sodium chloride in the diet, and more recently Luff has recommended the use of salts of potassium in preference to sodium on somewhat similar grounds. Clinical evidence generally hardly supports this view. By way of illustration consider the very large quantities of sodium chloride taken by patients at Weisbaden, where the main springs are notoriously rich in common salt. And there is no doubt that in the usual run of cases equally good results are obtained by treatment with these waters as with others in which sodium chloride or other salts of soda are only present in very sparing amount. Notwithstanding Roberts' elaborate researches we must regard the view of the existence of a quadriurate, more especially in the blood, as a pure hypothesis and not as a proven fact, and it is probably unwise to lay any stress on experimental observations on the "maturation" of the uric acid compounds outside the body as furnishing a basis for any deduction in the way of therapeutics.

Ord in 1872 advanced the view that inflammatory or degenerative changes in the affected tissues are to be regarded as the primary cause of gout, such initial changes not being caused by urates. He believed that gout was the sequel of a special form of degeneration in some of the fibroid tissues, resulting in an excessive formation of urate of soda, which passes into the circulation, and is later deposited in areas least freely supplied with vessels and lymphatics.

Sir Dyce Duckworth is the leading exponent of the view which regards gout as intimately connected with a disordered state of the central nervous system. He believes that, as a result of a disorder of some part of the neurotrophic system, a derangement of metabolism is induced which leads to undue formation of uric acid, and also inhibits the normal breaking down of that substance in the tissues, these things being accompanied in the case of acute gout by temporary weakness in the excretory capacity of the kidneys.

Kolisch in 1895 suggested that some antecedents of uric acid are the cause of the toxic effects which he believes constitute the primary cause of gout. This theory was mainly based on the presence of an increase in the alloxur substances (xanthin, etc.) in the urine of the gouty, but in view of the results of numerous investigations recorded in the last few years showing no such increase, this idea is no longer maintained.

A study of these various views shows how little we really know of the etiology of the disease. Garrod's great discovery was unfortunately followed by the result that all attention has been focussed on uric acid as the all-important factor, while in reality there is every reason to believe that it is by no means the main etiological factor, but only one of the incidents in the development of the disease.

The writer has lately described the appearance in the blood in gout of certain peculiar forms of leucocyte, which were considerably increased in number during the paroxysm. Whatever the nature of these cells may be, their occurrence lends force to the view that we must take a broader and much more general view of the disease than heretofore, and not restrict our investigation to any single line of inquiry.

With our present knowledge it is futile to theorise as to the etiology of the disease. All we can do, or at any rate all we ought to do, is to indicate clearly the known facts as to the state of the urine, blood, and tissues in the disease, and wait for the further advances in our knowledge of physiology that are absolutely essential for the elucidation of the problems presented by the tissues in gout (*see* "Uric Acid").

Before passing to a consideration of these special points, reference must be made to certain general facts of some importance in the etiology of the disease.

Sex and Age, etc.—The disease in its most characteristic forms is more common among men than women, but its various irregular and minor manifestations, especially those seen in hereditary cases, are at least as common in the female sex. When there is a strong hereditary history, typical joint attacks may occur in young men in their teens and onwards, but as a rule such attacks are more frequently seen at and about the fourth decade. In hereditary cases

various undoubted indications of a gouty tendency may be exhibited almost from infancy onwards; in other cases the first manifestation of the disease may develop in old age. With regard to bodily conformation it is usually supposed that persons of large frame and vigorous appetite, with a tendency to corpulence, are specially predisposed to gout. This is in the main true (such persons eat largely and often take little exercise), but at the same time many of the most typical gouty subjects are thin, pale, and dyspeptic-looking individuals.

Climate and Season.—Trousseau stated that one of his patients told him his joints were "barometers," and general clinical appearances at the present day afford not a few corroborative examples. The influence of meteorological conditions on general well-being (intra-cellular metabolism) is profound, but the nature of that influence is quite unknown. It has long been considered that gouty subjects fare better in an inland and hilly country than at the seaside, and there is a good deal of evidence in support. With regard to seasons, it is generally held that acute attacks of the disease occur with greatest frequency in the spring and autumn, but it is doubtful if this is really so to any noteworthy extent.

Drinking-Water.—It is probable that the drinking-water exercises a greater influence on gout than we know of at present. There is sufficient evidence to indicate that waters rich in lime are unsuitable, and there is little doubt that a combination of a clay soil and lime rich water are particularly injurious.

The Blood in Gout

Uric Acid.—The blood in gout contains an excess of uric acid. In well-marked cases, and especially during the paroxysm, this can readily be determined by Garrod's thread test, which is performed as follows:—To two drachms of serum obtained from a blister (applied at a site other than the inflamed spot) add ten to twelve drops of strong acetic acid. Mix the two fluids, and immerse one or two linen threads, and set aside for twenty-four hours. Then examine with a low power of the microscope, and if a positive result is attained, numerous minute rhombic crystals of uric acid will be found on the submerged part of the thread.

But the exact relationship between this increase of uric acid in the blood and the clinical features of the disease is by no means determined. One difficulty in determining this lies in the fact that uric acid is frequently present in considerable amount in the blood in conditions which have no known relationship to gout. Specially is this so in leucocythæmia, when there may be an amount of uric acid present in the blood far in excess of that seen in cases of gout, and yet there are present in these cases none of the clinical features either of acute, chronic, or irregular gout. Similarly with other morbid conditions, *e.g.* lead poisoning, although to a much less degree. The writer has recorded (*Brit. Med. Journ.* Jan. 28, 1899) the results of an investigation of the blood in cases of pneumonia, malignant disease, chronic Bright's disease, ulcerative endocarditis, and acute aneurysm, in all of which the presence of uric acid could be determined in the limited quantity of blood examined. Similar results have been found by other observers. Petren (*Archiv f. exp. Path. u. Pharm.* Bd. xli. 1898) found it present in a case of hysterical hæmatemesis and in one of gonorrhœal rheumatism, and he further emphasises its presence in anæmia as well as other diseases above mentioned. Weintraud also found that even in the healthy, uric acid can be demonstrated in the blood after the administration in very large amount of articles of diet rich in nucleins. These various points alone prove how inadequate the one factor of uric acid is in the etiology of the disease. Variations in the amount of uric acid in the blood clearly do not constitute the root evil, a fact emphasised by the point already noted, that Magnus Levy has shown that there is no appreciable difference in the amount of uric acid present in the blood during the acute attack compared to the free interval.

The Form in which the Uric Acid circulates.—This is in reality quite unknown. Sir W. Roberts' view has been already referred to, but as we know so little of the behaviour of the proteids, and also of the acid and alkaline salts of the blood in their relation to uric acid, we must regard his view as a hypothesis and not as a known fact.

Alkalinity of the Blood.—Much has been said and written about variations in the alkalinity of the blood being an important factor in the genesis of gouty symptoms. As with some other aspects of the disease, a good deal more is written on the subject than is warranted by facts. Reference has already been made to the importance of Magnus Levy's observations, as indicating that there is no appreciable difference in the reaction of the blood during the paroxysm compared to the free interval. Notwithstanding the writings of Haig and others, it may safely be asserted that there is no authoritative instance on record in which a diminution in the alkalinity of the blood has been clearly established (v. Noorden).

The Cellular Constituents.—The red blood corpuscles show no material change. The blood-plates exhibit no increase nor other definite abnormality, a point of some interest in connection with the undoubted liability to thrombosis in gouty cases. Balfour has indeed suggested that the phenomena of the acute attack may best be explained on the view of a local thrombosis. The only striking changes

observed have been in the leucocytes. The writer has recorded (*Brit. Med. Journ.* Jan. 6, 1900) the appearance of a peculiar type of white cell, large in size (15 μ), with a large oval or horseshoe-shaped nucleus, poor in chromatin, the protoplasm vacuolated and imperfectly stained.[1] These cells could be readily differentiated from the ordinary finely granular oxyphile leucocyte, and also from the lymphocyte; in form and general appearance they resemble degenerated myelocytes. During the acute attack these cells were considerably increased in number, and their presence suggested the possibility of their having an important relationship to the alterations in the uric acid and phosphoric acid excretion observed in the same case.

The results of a more recent investigation, in course of publication, showed the presence of somewhat similar cells, and their increase in number during the paroxysm was even more striking than in the former instance. This case is possibly of even greater interest, as it is more than possible that the acute attack was in great part induced by the artificial administration of nucleic acid, which was tried as a therapeutic agent. Much further investigation is necessary before we can speak definitely as to the significance of these cells, and in the meantime we may refrain from theorising regarding them. Their presence in the two cases, and the great increase in number during the acute attack, are, however, suggestive of their possessing considerable etiological significance.

The Urine in Gout

As we may look to the acute paroxysm to furnish us with the clue to the etiology of the disease, we will in the first place consider the condition of the urine during the acute attack. It is usually said that the urine then exhibits all the characters of a febrile urine, but this is by no means always the case. Transient albuminuria is present in a number of cases, especially in recurring paroxysms. The greatest interest, however, centres in the excretion of uric acid and other nitrogen-holding substances. There is now abundant evidence to show that during the attack there is not only no diminution in the amount of uric acid excreted, but there is frequently an actual increase, sometimes considerable. As already mentioned, Kolisch's view that during the paroxysm the alloxur bases are diminished has not been confirmed by other observers. We must also bear in mind that the laboratory difficulties in the investigation of the alloxur bases make their records less valuable than those of other substances.

Of greater importance is the excretion of total nitrogen. This is considerable during the attack, but this is doubtless in part due to the general febrile reaction. From the numerous detailed records available there is some evidence to show that for some time preceding the attack there is an actual retention of nitrogen in the tissues. The uric acid-urea ratio may be very little different from the normal, either during the attack or in the interval. With regard to the reaction of the urine very little can be said, except that we cannot lay stress on any apparent slight daily variations in its acidity. I have personally examined cases where the reaction was alkaline or neutral one day, and distinctly acid the next, and yet the amount of uric acid in the former was much less than that in the latter. Another point may be referred to. Attempts have lately been made to prove that the excretion of uric acid and phosphoric acid (two end products of nuclear disintegration) go hand in hand. It is very doubtful if this is the case. My own observations are strongly opposed to this view, and on other grounds one is inclined against it. It is true that uric acid and phosphoric acid are two end products of metabolism of the nucleins, but we must remember that the uric acid formed in the body is probably capable of further and ready transformation into urea, while there is nothing to indicate that phosphoric acid can undergo any further change in the economy.

As regards the urine in chronic gout, it is generally believed that the uric acid excretion is small in amount in the intervals. This may be so in some cases, but it is by no means invariably so. There is considerable individuality in each urine, and some of the worst cases show a very large amount of uric acid in the free interval. The occasional occurrence of glycosuria, phosphaturia, and oxaluria has already been referred to.

Conclusions

Do these findings in the blood and urine lead to any conclusions as to the etiology of the disease? We must unhesitatingly answer in the negative. At the same time, we will act wisely by refraining to formulate theoretical deductions. The generally accepted belief that the primary development in gout is the heaping up and deposit of uric acid in the tissues must be regarded as quite inadequate in view of the points referred to under the state of the blood. The relationship which the increase of uric acid in the blood bears to the local deposit is entirely unknown. Is there any actual increased formation of uric acid in gout? This query at once raises another, Where is uric acid normally produced? As the latter question has not yet received a final answer, we must speak with

[1] In some cases the films were fixed with heat (120° for fifteen minutes), and in others with alcohol and ether for an hour. Some were stained with eosin (2 per cent watery solution for two minutes) and hæmatoxylin (Hansen's solution, three minutes); others with eosin and methylene blue. In those fixed with heat special attention was paid to the granular leucocytes.

diffidence on the former. The older views, which regarded uric acid as an intermediate product in the combustion of nitrogenous substances into urea, have long been abandoned. The observations of Kossel, Mares, Horbaczewski, and others, referred to under "Uric Acid," have in the last ten years thrown much additional light on the subject. As a result of their investigations, the theory was advanced that uric acid was derived from *nucleins in the cell nuclein*, and that the excretion of uric acid bore a close relationship to the number of leucocytes in the blood. This theory has not been borne out by further recent investigations. The view was then advanced that the uric acid arose from decomposition of the *nucleins of the food*, and much discussion still rages on this point. Others consider that it is exclusively derived from the organised nuclein of nuclear disintegration, and attribute to the nuclein of the food an indirect action only, through an acceleration of metabolism and cell disintegration. There is no doubt that the administration of a diet rich in nucleins, *e.g.* thymus gland (sweetbread), leads to a very considerable increase in the excretion of uric acid, but the manner of interpreting this result is by no means clear. Many things have to be carefully considered. To illustrate: it is well known that in addition to uric acid other nitrogen-holding substances—so-called alloxur or purin bases—are excreted as the result of nuclear disintegration, but as our methods of determining these substances can hardly yet be said to be thoroughly trustworthy, conclusions arrived at from these investigations are not wholly reliable. Further, there is sufficient evidence to indicate that the organism of man can further transpose uric acid into urea, a fact of some importance in connection with views recently advanced as to the excretion of uric acid and phosphoric acid running a parallel course. These two substances are two end products of nuclear disintegration, and as there is no reason to believe that phosphoric acid undergoes any further change in the organism, while there is good reason to believe that uric acid can do so, it would be surprising if the excretion of the two substances went strictly hand in hand. Apart from this theoretical reasoning, my own investigations have convinced me that no such parallel exists. There is also no doubt that the *nucleins present in cell nuclein generally* are a source of uric acid production, but the conditions which influence the formation and decomposition of these nucleins in the body are quite unknown. Other writers maintain that individual dispositions vary considerably with regard to the amount of uric acid produced under apparently similar conditions. The whole subject of uric acid formation is still obscure, and it may well be, as Minkowski suggests, that the significance of nuclein-holding articles of diet may ultimately fall quite into the background. We must remember that the experiments recorded with thymus feeding cannot be regarded as comparable to the ordinary dietetic measures in daily use.

We are not in a position to make precise statements as to the conditions which influence the formation and excretion of uric acid in health, and still less are we qualified to speak of the state of affairs in gout. This being so, it is not surprising to find that the fundamental questions in the problem of gout are variously answered. One such question pertains to the kidneys. Are the kidneys primarily or mainly at fault in gout? This may be so; but if so, this has not to do with uric acid. Not only is the actual output of uric acid not diminished (often actually increased) during the acute paroxysm, but I have lately shown that the tissues of a gouty subject react like the normal tissues to the influence of pure nucleic acid (*Journ. of Path.* 1900), and Knoll had previously shown that they react like normal tissues to the influence of thymus feeding. In gout it is yet impossible to state whether we are dealing with an increased formation of uric acid, a diminished excretion, a diminished oxidation, an alteration in the solubility, or other unknown factors. Much further investigation is necessary before these points can be determined.

Finally, I will merely throw out a few suggestions which occur to me as having some importance in connection with the etiology. These remarks or queries will be based mainly on the following two points: (*a*) There is ample evidence to prove that the uric acid in the blood is not the primary factor in gout; and (*b*) uric acid can be deposited in cartilages and other tissues, even in considerable amount, without the association of any inflammatory phenomena.

(i.) The last-mentioned point clearly proves that the uric acid is not the factor which causes the inflammatory phenomena characteristic of the acute attack.

(ii.) What, then, are the toxic principles in the blood which possess the power of inducing the characteristic inflammation? and

(iii.) What are the factors which determine the incidence of these toxic substances from the blood into the tissues?

In connection with these queries we have to consider the all-important part played by the alimentary canal. Here we have, doubtless, one of the important keys to the solution of the problem.

(iv.) What effect has the absorption of the products of abnormal digestion of proteids on the blood? This abnormal digestion may arise from quite a number of circumstances, *e.g.* excess of proteid, faulty admixture of food stuffs, the effects of alcoholic stimulants, the result of fatigue from excessive muscular exercise, etc.

In this connection it is well to emphasise that this question will never be ascertained by mere analysis of the intestinal contents. What we desire to know is not so much the nature of the toxines as the reaction of the tissues—blood, blood glands, and tissues generally—to their influence. Yet another query.

(v.) Are the injurious effects of various carbohydrates, liquors, and other substances to be explained solely by their interference with the digestion of the proteids in the alimentary canal, or have they in addition the power of more directly exercising an injurious influence on the blood and tissues?

Heredity

The tendency to gout is inherited; it is not infrequently transmitted by the female line (cf. Chart), although specially manifested in males; in hereditary descent the tendency becomes inextricably mixed up with the tendency to other diseases — rheumatism, rheumatoid arthritis, tuberculosis, etc. If we add to these points the statement that environment, using that term in its widest sense, is an all-important factor in the explanation of the numerous peculiarities seen in hereditary transmission, we have stated all that is definitely known on the subject. Hutchinson and Duckworth accept the French view of the existence of an arthritic diathesis, from which arise as branches two distinct classes of disorders commonly recognised as gout and rheumatism. The former further agrees with the views of the older writers who recognised a mixed condition of the two disorders. There is every reason to believe that this mixed condition frequently occurs, but the term rheumatic gout is a misleading and inaccurate one. I do not intend to enter into any discussion as to the relationship of gout to rheumatism and rheumatoid arthritis, but refer the reader to the valuable writings of Hutchinson and Duckworth. While the subject is an interesting and fascinating one, our present knowledge will not admit of any practical observations on it. As Thomson has well said, the hopeful outlook is not in theorising but in experiment, in the collection of precisely observed data, and in the skilful use of statistical methods.

In gout certain experimental investigations are necessary before our knowledge of its hereditary aspects can be advanced. These can best be carried out by the induction of gout in lower animals, *e.g.* hens, etc., observing the effects of transmission of the gouty tendency to succeeding generations. At the same time, the statistical method is of undoubted value, but from its nature is clearly less valuable unless the statistics are complete and reliable, a combination not readily obtainable in practice. For some years I have been interested in carrying out this method as opportunity afforded, and have been fortunate in securing a particularly good illustration of the effects of the transmission of the disease. I am satisfied that the information given in the charts is reliable, but it is doubtless more incomplete than indicated. Nevertheless many points of the greatest interest can be learned from them. The relationship to rheumatism, diabetes, obesity, tuberculosis, malignant and other diseases, all find representation in a single family.

Gout is a general disorder of metabolism, and its adequate consideration requires careful study of the various derangements seen in succeeding generations, which in turn includes a consideration of variations in environment in the different individuals. With regard to one of the associated derangements of metabolism it is interesting to note that according to the Registrar-General's returns the mortality from diabetes is on the increase ("Diabetes," vol. ii.).

Diagnosis and Prognosis

The diagnosis of gout must be made from the history and general character of each case. In acute gout, and in chronic cases with well-marked external manifestations, there is, as a rule, no difficulty, but in many other cases there is no pathognomonic symptom whatever to distinguish it from rheumatism, with which it is liable to be confounded. The intermediate diagnosis of the two diseases approach each other so nearly, that to discriminate between them with our present knowledge may be impossible. In acute gout of the monarticular type a differential diagnosis has to be made from gonorrhœal arthritis. At least one case of very pronounced gout has come under my observation where the initial attack, which occurred at the age of twenty, and which involved the right wrist and back of hand, was erroneously regarded as a gonorrhœal arthritis. In this connection it may be well to state that Jonathan Hutchinson believes that gonorrhœal arthritis is more prone to occur in people of gouty habit, but in this instance that origin could be reasonably excluded, and the great rarity of the condition in a Scottish hospital fairly explains the error in diagnosis. In a few cases of acute polyarticular gout it may be by no means easy to differentiate acute rheumatism, and careful regard must be paid to the state of the blood, the previous history, and the measure of reaction to the remedies appropriate to each disease. Salicylate of soda in sufficient doses will certainly influence acute rheumatism, and will probably have very little or no effect on acute gout, while, on the other hand, colchicum will almost certainly exercise a remedial influence on the latter disease.

Although much could be written on the subject of diagnosis, much uncertainty must still attach to many instances of irregular gout, *e.g.* lumbago, sciatica, and the like. After all, the distinction is often of little practical importance,

as these cases are usually adults or older subjects, in whom the treatment of the individual case will not differ markedly to whichever genus of disease it may be assigned. (*See* also "Rheumatism.")

In a young subject affected with inflammatory or allied conditions in the hands or feet, or other irregular manifestations, especially if there be a distinct hereditary history, the possibility of the gouty nature of the affection should always be kept in mind, and while we must avoid the custom of attributing every morbid action occurring in a gouty habit to the influence of gout, we will be well advised in keeping in view the possibility of such a relationship.

Prognosis.—The prognosis of gout depends on so many circumstances, that it is hardly possible to speak with any precision on the point. Much depends on the age of the patient, the duration of the disease, the presence of complications, especially the state of the kidneys and cardiovascular system, and on the patient's will power and determination to give full effect to all appropriate treatment. Each case must be judged on its own merits, and if all these things are equal, which they seldom are, the prognosis, both as regards life and recurrence of the attacks, is eminently favourable. But we must admit that in a very small proportion of cases, in whom the disease develops early, and who give fairly full effect to appropriate treatment, there are a few apparently inexplicable recurrences of the disease. Such cases are, however, very exceptional; and the guarded prognosis, which is usually advisable, largely depends on the fact that the establishment of a rational and efficient prophylaxis is, from the nature of things, by no means readily attained. The prognosis from the point of view of life insurance will be referred to under that heading.

Treatment

General Considerations.—As the exact nature of the primary changes which lead to the uratic deposits characteristic of the fully developed disease is unknown, in the treatment we must be content with the adoption of those general and special measures which experience has proved to be beneficial in arresting the frequency and severity of their occurrence. These measures vary within wide limits. In the pre-uric-acid days the older writers regarded gout and plethora as practically synonymous, and they recognised two different types: one, the plethora of a healthy and vigorous habit, which is readily acknowledged and seldom misunderstood; the other, arising in a constitution more weakly by nature or depraved by vitiating influences, is characterised by gouty phenomena which are more liable to mislead. These clinical types still exist, and their careful study and appreciation will assist in arriving at the treatment appropriate in different cases. These remarks hold good for the fully-developed disease, but, as we have seen, we are much more frequently confronted with the treatment of minor and irregular symptoms, which develop early or late in life, and persist or frequently recur without ever culminating in the grosser manifestations. This, the early stage of the disease, is of the first importance. It is calculated to throw light on all the subsequent changes, and has neither received the attention to which it is eminently entitled, nor has it been given the place which it ought to have both in determining the true pathology of the disease and in establishing the treatment best suited to its relief (*vide* Mr. A., p. 516). In fine, the treatment of gout and the gouty diathesis is essentially a prophylactic one, and the results will depend on the length of time and the care with which the necessary prophylactic measures are carried out.

Of the treatment to be recommended and described, general measures relating to an all-round judicious mode of life are of much greater importance than any special medicinal or other remedies. A victim of gout soon learns that there is no radical remedy for his disease, and he sensibly holds himself aloof from all useless interference, relying more on careful regulation of his life, diet, and excretions. In arriving at a knowledge of the appropriate treatment, due regard must be paid to any peculiarity of constitution, whether natural or acquired, which may vary the aspect of the disease, and, further, the greatest assistance may be obtained from an observant patient, the facts so gained furnishing an important clue to the line of treatment to be followed in any given case.

While it is by no means determined that an increased uric-acid formation is a cause of the disease, there are sufficient indications to warrant us regarding a *diminution in the production of uric acid* as an important indication in treatment. This can best be obtained by, in the first place, regulating, *i.e.* diminishing, the total quantity of food consumed. The fact that through immoderate eating and drinking gout is favoured cannot be gainsaid, and while it is true that the disease may develop in people who are poorly and inadequately nourished, this fact merely proves that other influences may induce a like result. We must clearly distinguish between absolute and relative excess of food. By the former is meant that which would be excessive in the most healthy state of the individual constitution; by the latter we mean that which, although it may not exceed what the individual in a state of health, with moderate exercise, might safely indulge in, is yet relatively excessive when health has from any cause deteriorated. So long as the excesses are casual and inconsiderable, the self-adjusting powers of the body are sufficient to dispose of them; but when, from extent or continuance,

the excesses strain these powers beyond a certain point, the corrective energies of nature fail, and the outward and visible evidences of the disease result. In such cases well-marked constitutional disturbance had existed without the individual having any consciousness of its presence, which fact indicates that a decision as to the state of health of a typically gouty subject cannot be arrived at from the evidence of his own consciousness. There is also sufficient evidence to justify *a limitation in the articles of diet rich in nucleins*, *e.g.* thymus, liver, kidneys, but, as these form a very small part of ordinary feeding, they can, except in special cases, be overlooked.

If these points are attended to, a further indication in treatment is at the same time fulfilled, *i.e. the elimination of the uric acid.* It is well known that defects in the renal functions play an important part in the disease, and, if the strain on the kidney is lessened through administration for a short or long period of a reduced nitrogenous dietary, these organs will be enabled to cope more successfully with the elimination of uric acid and other toxic substances. This object will, of course, be more readily obtained if the assistance of the correlated excretory organs, the skin and intestinal canal, is secured and maintained.

Allusion has already been made to the older views on plethora, especially that arising from excess of nutriment, so-called nutritive plethora, the treatment of which mainly resolved itself into a limitation of the amount of food consumed. But the older physicians also recognised an "excrementitious plethora," usually a sequel of the other, with resulting far-reaching injurious effects on all the organs and tissues of the body. This condition suggested to them the advisability of closer attention being directed to the excretory function of the skin by means of baths of various kinds. Since that time Balneology has developed extensively, and has rightly come to occupy a very important place in the therapeutics of the disease. The action of baths in general on the heat-regulating function, on local and general tissue metabolism, on the general circulation, and on the central nervous system, are elsewhere described (*v.* Balneology), and need not be considered in detail. It must suffice to indicate the very important part which the skin can play as an organ of excretion of nitrogenous waste products (urea). This fact, of which there is abundant clinical evidence, is one which is insufficiently appreciated in most text-books on physiology, and the practical deductions to be drawn from it are too much ignored by the practitioner.

An interesting question relates to the subject of *uric acid solvents.* If we can so influence the blood and tissue fluids as to increase the solubility of the uric acid compounds, and thus facilitate their elimination, it is obvious that this will be one of the outstanding indications in treatment. As on many other points, opinions vary both as to the theoretical and practical value of these substances, but it is on the whole doubtful, if by the administration of any alkaline salts, the remarkably stable blood and tissue fluids can be so influenced as to lead to their exercising a solvent action on the deposit. In the absence of further proof, it is probably safer to conclude that any beneficial action possessed by these substances is primarily due to their diuretic action. The same statement can be applied to the use of the various organic nitrogen-holding bases — piperazin, lysidin, urotropin, etc.

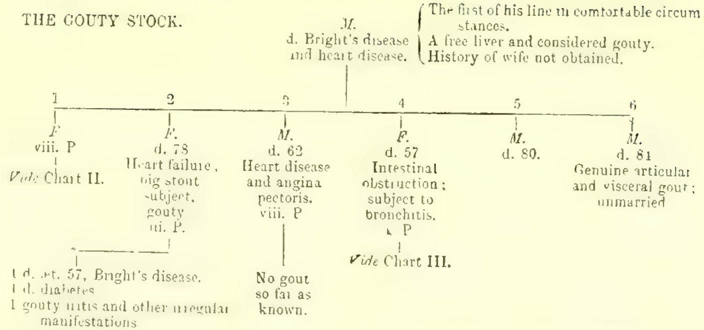
A point of greater importance, and of even greater difficulty, is the influence of the preformed salts in the different articles of food on the solubility of the uric acid. Here, again, we are on unknown ground, and if we are not in a position to speak definitely regarding the influence of the preformed salts of the animal and vegetable foods, still less are we able to speak of the action of the salts formed in the body from oxidation processes. The sulphur and phosphorus of the albuminous food-stuffs are oxidised into their respective acids; these cannot exist as such, and are immediately changed into various compounds; but the nature of these changes, either in healthy or deranged metabolism, is not determined. Similarly, while the organic acid salts present in vegetables are changed into alkaline carbonates, we do not know what influence they exert on uric acid compounds in the blood or tissues, and it is probably inadvisable to draw any conclusions from the solvent power of these and other substances *in vitro* applicable to the state of affairs in the living body.

We are on safer ground when we pass to consider *the beneficial influence of exercise* on the gouty subject. Everything which favours the inner or tissue respiration in the muscular and other tissues directly or indirectly fulfils the preceding indications, and in this category work and physical exercises of different kinds take a first place. These exercises should preferably be conducted in the open air, and should be of a kind adapted to the age and sex of the individual subject. Active walking, golfing, cycling, horse exercise, hill-climbing, and other seasonable exercises occupy a foremost place in the list. With regard to the extent to which these are to be advocated no hard and fast line can be drawn; the habit of body of each individual, and the state of the cardio-vascular system, must be carefully considered in each. Needless to say, the presence of organic disease in the kidneys, heart, or other organ will modify the extent to which these are to be indulged in.

While it must not be forgotten that in a few cases active exercise seems to have a prejudicial influence in inducing an acute attack, this should

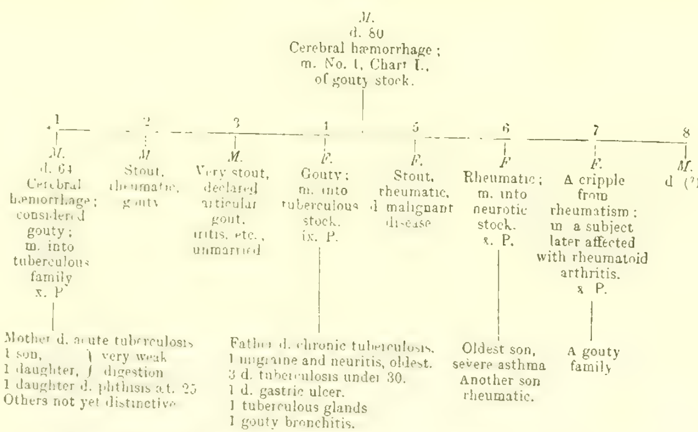
THE GOUTY STOCK.

CHART I.



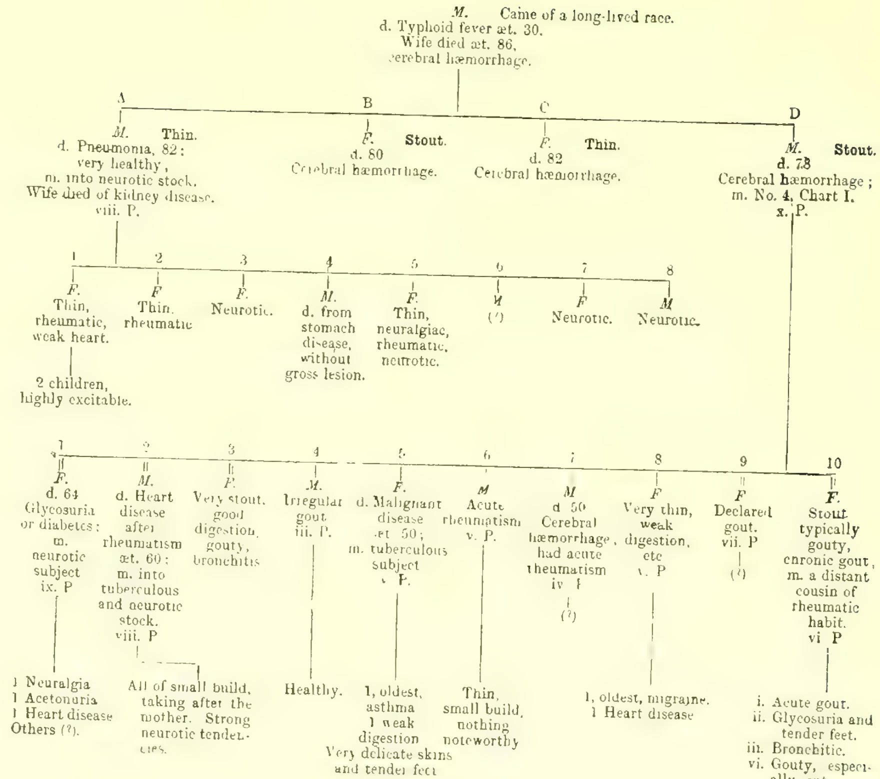
NOTE.—In the opinion of the family the father acquired gout through port wine and other proclivities. The youngest son was a martyr to the declared disease. The history of the daughters is specially interesting. Nos. 1 and 4 married cousins, and the diathesis of their children is seen in Charts II. and III. Note that the descendants of No. 2 are of the same generation as those marked * in the other charts.

CHART II.



NOTES.—The exact sequence of seniority in the above may not be perfectly accurate. The eight members were on the whole of a big and heavy build. Of the three sons whose history is known, one has typical gout and the others are gouty, one having died at 64 of cerebral hæmorrhage. Of the four daughters, one died of malignant disease, and all of them are rheumatic or gouty. The known existence of the latter strain leads the patients to lay much stress on that factor. Nos. 1 and 4 illustrate a blend with tubercle, the former being [illegible] circumstances, and the latter not so. Compare the progeny of the two cases, and note that in the latter the liability to tuberculosis is the preponderating influence, probably due to two factors: (a) general infection from the father's chronic condition, and (b) the absence of the exciting dietetic causes of gout. The occurrence of asthma in No. 6 is also of interest. The parents of No. 7 were both seriously affected only in late life, and their family are almost all of plethoric type, and some of them are known to suffer from irregular gouty manifestations. Their future history should be of great interest.

CHART III.



NOTES

i. In the light of Moore's observations as to the very frequent presence of urate of soda in the joints of those patients who die of cerebral hæmorrhage, Cases A, B, C, and D are of considerable interest. Their longevity is noteworthy, and so also are the two different types of bodily conformation.

ii. The continuation of the history of Mr. A. shows the great influence of the mother on the progeny. The neurotic strain is the prevailing one (Case IV. died when young from some gastro-intestinal neurosis, a pathological examination revealing no gross lesion); some of the family also regard themselves as rheumatic.

iii. With regard to Mr. D., while noting the original tendency to longevity, observe the apparent effect of admixture with a known gouty stock. Nos. 1, 2, 5, and 7 have died at an average of 56; note the history of each.

iv. In this large family the different types of bodily conformation, noted in a previous generation, are very marked—three are very thin, six are stout, and at least one is obese. The stout members have a double line.

v. A careful comparison should be made of all the families of the same generation marked with an asterisk. These are the children of three sisters.

vi. Cases 2 and 5 are of special interest as illustrating a blend with tubercle. In the former case the subject was not *herself* tuberculous, but most of her family had died from tuberculosis; in the latter the patient was *herself* tuberculous. The history of these cases should be compared with the similar ones in Chart II. In the cases under consideration the comfortable circumstances of the individuals were well calculated to foster a gouty tendency. Taken collectively they illustrate the all-important influence of environment on disease.

vii. No. 10 illustrates the effects of a blending of a rheumatic and gouty strain—an intensification of the gouty tendency.

in no way interfere with the general application of this principle. More harm results from too little than from too much exercise, and we should remember that the name of exercise is often given to bodily exertion so gentle that no increased activity of inner respiration results. When time and opportunities do not allow sufficient exercise in the open air, a fair substitute is found in the various available forms of home gymnastics, and in many cases a judicious combination of the two is very desirable. In all cases attention should be directed to those groups of muscles which, from the patient's occupation or disposition, are apt to be largely or entirely neglected.

Still more active measures are frequently advisable in cases with chronic joint affections, or in the case of involvement of fascia or local muscular and connective tissues. Such cases may be much benefited by various forms of active and passive movements. The benefit to be derived from this therapeutic measure may be emphasised by the fact that not a few typically gouty (usually irregular gout) adult subjects, men and women, regularly undergo, of their own accord, a course of medical gymnastics in one of the numerous Swedish mechanical institutes available for this purpose at the various foreign spas.

A further general consideration requiring attention is the *state of the central nervous system.* While the importance of this factor in this, as in most other diseases, is undoubted, there is, I think, a tendency to lay undue stress on it. While it is true that recurring indications of declared gout not frequently appear to be dependent on nervous causes, in so far as its manifestations declare themselves after some mental perturbation, typical attacks of acute gout occur with sufficient frequency, both in elderly and younger subjects, quite independently of any such exciting cause to justify us not laying very great stress on the relationship. Further, most of the undoubtedly gouty developments in young subjects arise and recur apparently independent of any direct nervous influence. On this ground, also, we are led not to lay very great stress on the nervous system as *the factor* of primary importance. Its ultimate importance cannot be gainsaid. It is one of the factors which continually varies the aspects of the disease.

Derangements in the digestive system play an exceedingly important rôle in the development both of acute and chronic gout, and these derangements usually depend either on immoderate eating or drinking, or on the ingestion of articles of diet especially unsuited to the individual. The probable relationship of these digestive disturbances to the phenomena of acute and chronic gout has already been referred to, but be that what it may, the importance of a well-regulated and healthy intestinal mucous membrane cannot be over-estimated. Auto-intoxication is certainly a primary factor in the disease, and has to be guarded against by careful dieting, healthy intestinal secretion, and normal intestinal evacuation. There is no doubt that in a certain proportion of cases a hydragogue cathartic, taken on the first indication of impending trouble, will avert the acute attack, and it is advisable to insist at all times on the closest attention being paid to the action of the bowels. While we have yet to learn much about the secretory or excretory influence of the large intestine, we may safely assume that by judicious purgation we do much more than merely free the system of the undigested food-stuffs present in the gut. The vigour of the constitution must be the standard by which the depletion is regulated, and where such vigour is deficient, measures of the same activity should not be pursued as are necessary when the system is robust. Not infrequently in the course of subacute or chronic gout there is developed a subacute or chronic catarrh of the gastric and intestinal mucosa, which in turn aggravates and complicates the general gouty state. Such a complication merely leads to a more strict application of the general principles of the dietetic treatment appropriate to the disease.

Dietetic Treatment

It is sometimes maintained that the influence of different food ingredients on the gouty constitution has been too much magnified, this assertion being based on two clinical facts—firstly, that a child fed entirely on milk may continue to excrete large quantities of uric acid ; and, secondly, that an adult patient who has been excreting a similar excess on a light vegetarian diet may speedily improve and return to a normal excretion when his diet is changed to meat. This view is based on too narrow a conception of the problems. The little we know with certainty regarding gout seems to indicate that the radical defect is in the metabolism of the proteids of the food. The metabolic changes of the proteids of milk or vegetables are essentially similar to those in meat, and we have no reason to believe that the decomposition products arising from the normal metabolism of the one are in any way different from those in the other. Any differences there may be are those of degree and not of kind. Everything depends on the form in which the food-stuffs are presented. It sometimes strikes me that in this question of feeding we find an interesting analogy in the art of agriculture. A scientific farmer in the feeding of his land has not only to consider the natural quality of the soil, but also the climate, rainfall, and nature of the product desired. Of the food required by the growing plant the element nitrogen is also one of the mainstays, and it is usually provided in one of

two forms—potassium nitrate or ammonium sulphate. Now it is by no means immaterial to him in which of these forms the land is fed. His choice depends on many things, one of the most important from our present point of view being their relative solubility. If the land is badly drained, and especially if the rainfall be considerable, he uses the less soluble ammonium sulphate in order to minimise the risk of the nitrogen being washed away and so rendered unavailable for the growth and maintenance of his grain. So it is with the human subject, but only in a more elaborate way. The individual qualities of the tissues, their drainage system and the relative solubilities of the different food ingredients as commonly prepared, constantly demand careful consideration. The question of diet is certainly the paramount one. Its influence is profound, not only on the individual, but on the race, and its importance was well defined by Sir William Roberts when he wrote that one generation of scientific dietetics would produce an influence upon humanity second only to a new creation of the race.

Meats, etc.—From the earliest times some writers have regarded all or at any rate most kinds of muscle food as injurious to the gouty, and as in other debated points in the subject history of the disease many experimental observations have been brought forward to prove this point, but, as a rule, these observations have been made in the much too narrow field of uric acid excretions. The most recent observations (Taylor) indicate that the commonly accepted view that a meat diet is associated with an increase in the uric acid excretion is an erroneous one, and on the whole we may take it as definitely shown, both by practical experience and theoretical experiments, that a gouty subject may take a measured quantity of meats in an easily digested form.

In the use of meats it is not only important that these should be taken in an easily assimilable form, but that they should not be accompanied by an undue admixture of other foodstuffs. It is held by some that in such cases the fact of the carbohydrates and fats being more readily oxidised in the tissues, leads to defective combustion of the albuminous foods. This subject has been previously discussed, but whatever the scientific truth may be, the fact remains that we must look for the cause of any injurious effects of meat more in its quality and in the form in which it is administered. If too little nitrogenous food be taken an increased decomposition of nuclear-holding tissues may result, as has been proved in a case of complete starvation, when typical acute gout developed in the course of the observation.

We must, however, bear in mind that a strong meat diet, that is, meat twice or thrice daily, is an acid food owing to the imperfect neutralising of the sulphuric and phosphoric acid previously referred to. This may be in part rectified by the consumption of the alkaline table waters referred to later.

We have also to consider that the tastes and inclinations of the greater number of gouty subjects demand a certain supply of meat, and, by taking it in moderate amount, the supply of nitrogenous food necessary for the maintenance of the albumin in the body is more readily obtained.

With regard to the different kinds of animal food, white meats, *e.g.* fish and chicken, are more suitable than red meats owing to their more ready digestibility, and also in the case of fish to the smaller proportion of nitrogen present in equal bulk. The confirmed gouty subject is wise to limit his consumption of red meat to one meal in the day or even less, and further, to make as a routine a selection of the red and white meats similar to that indicated in case Mrs. E., p. 520. The whole question of a meat diet is summed up in its digestibility, which in turn bears a definite ratio to the simplicity of the meal in which the meat is a component part. The temporary diet of meat and hot water which is of such value in suitable cases, is a simple one, and to its simplicity we must largely look for an explanation of its beneficial effects. What has been said of meat holds also good for other animal foods. The various kinds of fishes and game can all be taken by a gouty subject, but what we must have regard to is the amount of admixture with other foods and drinks. Not infrequently innocent substances taken at the end of a highly nitrogenous mixed meal are regarded as the noxious agents when in reality they have played a quite subordinate rôle. High game and very fatty meats should be avoided.

Under this heading a few practical points may be mentioned about the culinary aspects of soups, meats, and fish. It is impossible to over-estimate the importance of this subject; the want of recognition of its importance is, I think, one of the causes of the very diverse differences of opinion entertained as to the beneficial or noxious influence of various dietetic substances.

Soups. What soups may a gouty person partake of?

The answer to this question will depend on the digestive capacity, but in coming to a decision we must take into consideration some elementary points in the preparation of the various soups.

It has been stated that there are perhaps not less than 500 soups, but regarded analytically, there are only a few leading species from which the different varieties are produced by additions and combinations of flavours.

(*a*) A clear decoction of meat or bones which, when weak, forms a broth or a "tea," and when

strong is a consommé or essence. These may be prepared from beef, veal, or mutton, and sometimes pork or ham.

(*b*) A similar decoction can be made from the various forms of fowl, game, and fish.

(*c*) A decoction of vegetables, including herbs, roots, grains, and farinaceous substances.

All of these soups may have added to them well-known dried Italian pastes, *e.g.* vermicelli or macaroni, and a consommé of meat or pork or game may be thickened with additions of a meat, fowl, or game purée respectively. In the same way the weaker broths furnish a basis for vegetable purées.

The following highly nitrogenous soups are not as a rule suitable articles of diet for the gouty :—Turtle, mock turtle, hare, kidney, ox-tail, mullagatawny. If they are indulged in, the rest of the food and drink consumed at the same meal must be more carefully limited.

Cock-a-leekie, giblet, and hotch-potch are almost stews, and should be considered as a meat course. In not a few cases all of these highly nitrogenous soups are contra-indicated.

The great fault to be found with soups usually served is that they are heavy, and contain too many ingredients. The average soup is made up with as many good things as possible, some to make it more nourishing, others to make it more palatable. This is all very well for the healthy, but where, as in gout, the digestive functions in the tissues and alimentary canal are readily disturbed, simple soups are required. The soups without thickening are therefore the most suitable. The thickenings in common use are purées of meat or fowl, flour, tapioca, yolks of eggs, etc.

A good rule in making soup is always to make it the day before it is required, as it has then time to cool, and the fat having risen to the surface can be easily and thoroughly removed. Soup should never be allowed to cool in the saucepan in which it has been made, but should always be poured into a basin, as otherwise the temptation to reheat without removing the fat is too great for most cooks.

Roast beef and mutton bones boiled with vegetables, and the fat carefully removed, make a good stock from which soup can be made for the gouty. Excellent soup can also be made from the water in which meat or fish has been boiled.

The various vegetable purées—spinach, artichoke, tomato, carrots, green peas, etc.—are excellent for this class of patient. They are sufficiently sustaining to prevent a feeling of hunger, and if well digested give a fair amount of nourishment. In the case of soups made from the pulses, their high nutritive value should influence the rest of the meal, and in some cases they are better avoided. A very good vegetarian stock can be made by extracting the "goodness" and flavouring from vegetables, the chief ones being onion, celery, carrot, and turnips. To do this take these vegetables and cut them into small pieces, place in a saucepan with sufficient water to cover them, and let them boil gently for several hours. The liquor when strained off is "stock," and can be flavoured with a small quantity of savoury herbs, pepper, salt, and ketchup, and can be coloured a nice brown with a few drops of Parisian essence.

A more detailed account of the methods of preparing the invalid soups (teas and essences) will be found under the heading "Invalid Feeding."

Meats.—Made-up meats are not suitable for the gouty, owing to the greater toughness of fibres induced by the second cooking, and also because of the admixture of rich sauces of various kinds which are usually added for palatability. Meats should be tender and simply prepared. The best ways of preparing are in order—broiling, steaming, roasting, boiling, baking, stewing, and frying. The last mentioned should be avoided, especially in the case of beef and mutton. Although lamb and veal possess less extractive value than other meats, and are on that ground commendable, yet the gelatinous nature of the fibres makes them more difficult of mastication, and therefore less digestive. If allowed, this danger must be pointed out with the view of obviating it. Tripe, sweetbreads, kidney, and liver may all be allowed for occasional use, provided the very special cleaning and careful cooking necessary are given effect to. Salted meats are rendered more indigestible in the preparation, and should therefore be avoided. Bacon and ham are more digestible than pork. With regard to game, white flesh is more suitable than brown, and water-birds are more fatty than other game.

Fish.—Fish are well calculated to form a large proportion of the dietary of the gouty. They contain on an average one-third less nitrogen than an equivalent amount of ordinary meat, and usually contain little or no fat. Fat fishes (salmon, mackerel, eels, pilchards, red mullets) are equal in nitrogenous value to an equal amount of moderate fat beef. There is a very large proportion of water in the flesh of fish. When ordering, it is well to suggest those known to be in season and plentiful, as they will probably be in the best condition. As has been already mentioned, many nutritious soups can be made from them.

Whiting, smelt, and sole are the most delicate and easy of digestion. Haddock, flounder, grey mullet, and plaice, are also good ; the haddock has more flavour, but a coarser grain than the whiting. Mullet and halibut have firmer fibre, and are better cooked in large pieces. The fatty fish referred to are very tasty, but are more prone to derange digestion. In the salmon

the fat is mainly found on the underside, and a slice from the back may be readily taken when the underside disagrees. Cod, unless in the very best of condition, is the least digestible of all white fish owing to the great toughness of its fibres, but the head, when boiled, makes an excellent stock for soup. Of the shell-fish, oysters, mussels, and scallop are all good, and so are also lobsters, crayfish, shrimp, crab, and prawn. Lobster salad or crab pie is quite allowable for young subjects with good digestion. Its nutritive value is not high, and its acceptability to the palate is beyond question. Fish should never be over-cooked. The best methods of preparation are boiling and steaming, then comes broiling and grilling, and lastly frying. In not a few cases we find fish are not permissible. Much, however, depends on the cuisinère. A combination of curried fish and rice makes a good dish. When digestion is obviously weak sauces are better avoided. One of the simplest is a little melted butter with chopped parsley. The familiar "butter sauce" is usually an imperfectly cooked indigestible compound.

Meat and Hot Water Cure.—Before leaving the subject of meat reference may be made to the value of an exclusively red meat diet in certain cases of chronic gout. Armstrong (*Med. Soc. Transactions*, 1897) has recorded a series of interesting cases, in which after a course of the usual routine treatment had failed, very great benefit was obtained by the so-called "Salisbury" treatment. The essentials of this treatment are the drinking of three to four pints of hot water daily, a pint to be taken before each meal, and the same quantity at bedtime, and the administration of one to five pounds of meat in the twenty-four hours. The meat should consist of beef-steak freed from fat, gristle, and connective tissue, well minced, a little water being added, and then warmed through with gentle heat. I have had no personal experience with this treatment, but can readily accept the moderate claims made for it in the paper referred to. The probable explanations of its value have been previously discussed. I would just like to add from experience in other cases that if the meat as thus prepared is not readily digested, a yet simpler and more digestible form may be found by taking the best beef-steak, freed from fat, and rubbing through a fine sieve until the juice has all passed through, and all the fibrous parts remain. The mince should then be treated as above. The appearance when cooked is just like very fine mince. The value of this preparation is by no means confined to gouty dyspeptics. The course of treatment lasts from four to twelve weeks, and thereafter the patient makes a gradual return to an ordinary dietary. The conclusions formulated by Armstrong from his experience of the treatment are as follows:—

1. That a certain number of cases (not more than 3 per cent) of chronic gouty arthritis, recurrent uric acid calculi, and gouty dyspepsia, with fermentative changes, which have proved refractory to ordinary methods of treatment and dietary, may be treated by means of an exclusively red meat dietary, plus hot water drinking, with excellent results.

2. That this method of treatment is irksome and trying, and as, unless it is carried out *strictly* in the first instance, it is apt to do harm, it should only be used in those cases where other methods have failed, or are thought likely to do so.

3. That the cases require careful selection and close medical supervision, the details being modified according to the needs of each individual patient.

4 That those who suffer from persistent albuminuria or organic heart disease are in most instances unfit for this treatment; when, however, it is prescribed for them its course should be watched daily.

5. That certain cases of chronic gouty arthritis which fail to improve while on a mixed diet, recover equally well whether on this dietary or on the meat free dietary suggested by Dr. Haig.

6. That it is of the utmost importance that no addition, however small, of carbohydrates, saccharine matters, or fruit be made to the dietary during the first few weeks of treatment—very slight acts of carelessness in this respect having often caused disappointment and failure.

7. That used with due care and discretion this method is a most efficient and sometimes even a brilliant addition to our therapeutic measures.

The reader is referred to the original paper for a more detailed account of the treatment and of the reaction of the tissues under its influence.

Milk and Milk Products.—As in the case of various other foods, there is much difference of opinion as to the value or necessity of a diet composed largely of milk, milk products, and vegetables. In many cases a course of a strict milk diet is the most suitable, particularly in young and otherwise healthy subjects, but it is much less suitable for adults and the aged. Milk is highly nutritious, and when it is easily digested and no undue fermentation processes induced by its use, a limited course of milk diet is to be commended, the amount and duration being regulated by the effects on the digestive system and by the attitude of the patient towards it. Alkalinity of the urine is favoured or increased by a full milk diet, yet an exclusively milk regime is probably in the main unfavourable. However, there is no doubt that the children of gouty parents should be brought up systematically on a diet in which milk and its products are the staple, and meat given in very limited quantities. This is specially im-

portant in those by no means infrequent cases where there is, in addition, a marked neurotic strain in the family (*vide* "Adolescent Insanity"). With the active and fixed habits of later life a meat-free diet is very rarely practicable, and is very seldom called for.

Cream, forming as it does the most appropriate form of fatty food in the dietary of the aged, likewise constitutes an excellent form of fat administration in gouty subjects. It should preferably be taken with milk pudding or stewed fruit in an otherwise simple meal, or it may be used in the preparation of chicken cream, fish cream, or in various combinations with vegetables, when it takes the place of butter (*vide* "Invalid Cookery"). Cream which has not been treated by any artificial process is always to be commended.

Skim-milk is more digestible than ordinary milk in all cases where fat is not readily digested, but in recommending it as a beverage or food regard must be paid to the amount of proteids and lactose present in it.

Whey is a useful article in many cases. It is a pleasant and stimulating drink, with a certain food value from the lactalbumin, lactose, and mineral matter present. In some cases whey plus cream make an admirable combination.

The pure caseinogen of milk, now prepared by the Protean Company, in the form of a flour, and made into biscuits and bread, is an appropriate form of proteid administration. The taste, however, is an acquired one.

Cheese.—There is no reason why cheese should be forbidden. The ill effects frequently attributed to it arise from the manner in which it is taken at the end of a meal, already excessive and badly assorted. Being a rich albuminous food, and varying in the proportion of fat present according to the variety of cheese, it should not be taken in large quantity; it should be well masticated, and it should be carefully distributed through the various vegetables or bread-stuffs of the meal (*vide* p. 521).

The composition of cheese depends entirely on the amount of fat it contains. There are three leading varieties—soft, hard, and skim-milk. A fair average for the fat and nitrogen present in these three types, *when fresh*, is as follows (Fleischman):—

	Soft.	Hard.	Skim-milk.
Fat . .	31 to 44	29	2 to 3
Nitrogen .	13 to 24	28	19 to 33

The fatty acids present in cheese have been considered detrimental, but, on the other hand, it is possible that in some cheeses, especially the richer varieties, the acids formed by bacteria in them may be inimical to other putrefactive processes going on in the alimentary canal. If so, this would explain the absence of injurious effects from the taking of these cheeses noted in some cases of otherwise very weak digestion.

The soft cheeses include cream cheese, Wiltshire, Neufchatel, Gorgonzolas, and numerous other Italian and French cheeses. The hard cheeses, which ripen slowly, and are adapted for keeping, include the English and American Cheddar, Stilton, Cheshire, Gloucester, Dunlop, Parmesan, Gruyère, and most English, Dutch, and American cheeses. Gouda and Raden may be taken as illustrative of the skim-milk variety.

It is well to recommend patients who are very fond of cheese to partake of one of the softer varieties, as, although less digestible, they are much less likely to be taken to excess.

Eggs.—Eggs are an excellent dish for the gouty, and should form one of the staple breakfast dishes. They also constitute a very appropriate food constituent for the children of gouty parents, in whom the consumption of meat, and especially red meats, should be very limited indeed. It is well to remember that a hard-boiled egg takes three hours, and a soft boiled or raw one, from $1\frac{1}{2}$ to 2 hours for complete digestion.

Carbohydrates.—The fact that gout is unknown in countries like Japan, where a strict vegetarian diet is common, clearly proves that, as a class, carbohydrates can by no means be the direct cause of the disturbance of metabolism characteristic of gout. On the other hand, taken in considerable amount and with strong nitrogenous food-stuffs, their use is frequently accompanied by some evidence of local disturbance in the alimentary canal, or general disturbance of metabolism in the tissues, either of which may be characteristic of a gouty proclivity of tissue. When this tendency is pronounced, the sum of the local and general effects produces typical gout in a more or less acute form, the manifestations depending on the age and constitution of the individual. In other cases we find irregular gout, which, when well marked, may be regarded as equally typical, as it is dependent on internal peculiarity of tissue.

Carbohydrates, and especially those of the saccharine group, are as a class to be regarded as more potent noxious agents than meat. The explanation of this is probably found in not one, but many factors, which have already been alluded to under Etiology. A good rule with regard to them is to reduce the amount and simplify their form.

Saccharine foods and dietetic accessories, *e.g.* jams, marmalade, sugar, sweet cakes, are only to be partaken of occasionally and in small quantity, and in not a few cases, especially of stout adults, are to be studiously avoided.

With regard to the strict vegetarian diet so eloquently advocated by Haig and others, the good effects undoubtedly derived in many instances depend, in my opinion, on the simplicity of the whole diet, with the limited quantity of the chief nitrogenous ingredients, these being the two primary essentials in the

dietetic treatment of confirmed gout. The following illustration may be given (Haig):—

Breakfast.

1 pint of milk.
Bananas, Apples, Pears, Plums, fresh, dried or cooked, Any other fresh fruit — *ad libitum.*

Lunch.

Vegetable soup made with milk.
Plate of potatoes (with butter, oil, or milk).
2 oz. cheese, eaten with potatoes and any other vegetables in season.
Stewed fruit or tart.
Fresh fruit.
1 pint of milk drunk during the meal.

Dinner.

Much as lunch.
1 pint of milk.
1 oz. cheese.

A close analysis of this diet, which is recommended for a person in health, shows that it is not quite so simple as at first sight apparent, and while a diet for the gouty framed on very similar lines is undoubtedly a very beneficial method of treatment in some cases, in others it is altogether unsuitable.

Popular belief, partly supported by medical opinion, condemns potatoes, but if used in moderation, and cooked and served with due precaution, there is no reason for prohibiting them, except in those special cases where they are definitely determined to be unsuited to the digestive capacity.

When new and moist they are indigestible; the best form is a well-boiled mealy potato in its skin, or the same put through a potato masher. A thoroughly well-baked potato is also good. When fried, or roasted in mutton dripping, or mashed with milk and butter, they are unsuitable in most cases. The other roots—turnips, carrots, parsnips, radishes, beetroots (also rich in sugar), artichokes, also cabbages, curly greens, Brussels sprouts, broccoli, and the green of cauliflower—should only be taken in small amount on account of their tendency to induce flatulence, etc. The following are more suitable: Spinach, flower of cauliflower, savoys, endive, lettuce, watercress, kale, leeks, onions, celery, cucumber, vegetable marrow, green peas, French beans. Asparagus has been condemned by some writers on account of the nucleins in the young shoots; also tomatoes and sorrel on account of the acids present; but, as many typically gouty subjects can partake of them freely, it is probable that these objections have only theoretical importance.

The green vegetables above mentioned can be freely partaken of in the form of salads, provided oily dressings and hard boiled eggs are avoided.

Mushrooms and truffles and other fungi are quite permissible in small quantities.

The pulses (lentils, peas, beans, haricot beans) are not as a rule advisable, because it is not an easy matter to make the patient realise that their nutritive value is such that their use must influence markedly the amount and quality of the other articles consumed. With regard to *puddings*, the simpler the better. Milk puddings, such as rice, sago, semolina, ground rice, etc., should be made without eggs in many cases. Suet puddings of all kinds are as a class to be avoided, but if made with bread-crumbs in place of flour, well boiled and unaccompanied by a heavy sauce, they may be taken without prejudice. If custards and omelettes, sweet or savoury, are taken, the nutritive value of the eggs must be recognised. Jellies, blanc-manges, lemon sponge, and creams may be taken in very sparing amount, and, as with other foods, a wise discretion is necessary both by the physician and by the patient. Fruits of all kinds in themselves are permissible, but must be taken with caution, especially in later adult life, and it is well to bear in mind the old saying, "Fruit is golden in the morning, silver at midday, and lead at night." Much depends on the amount of sugar used in the cooking and the accessories used at table.

Beverages

To the confirmed gouty subject the question of what he may drink is sometimes a more important one than that of what he may eat. Alongside of this question there is another bearing on the time—relationship to meals—at which various liquids should or should not be taken. Thus, it is as a general rule advisable to recommend alcoholic stimulants only to be taken with meals; in other cases the consumption of fluids, of a non-alcoholic nature, may be wisely restricted mainly to the intervals between meals. Fluids may act prejudicially in two ways. In the first place, they may act injuriously in a mechanical way, *i.e.* by clogging the food elements, and at the same time diluting the digestive fluids, favour abnormal decomposition of the proteids and also carbohydrates. And, secondly, they may, in virtue of a specific action, lead to faulty nitrogenous metabolism in the digestive tract, and secondarily, in the tissues.

Like the question of diet it is impossible to lay down definite rules applicable to the disease. Everything depends on the age of the patient, his constitution, his previous history as to consumption of fluids of different kinds, the nature of the symptoms and the reaction of the tissues to various fluids. Specially is this so with the use of alcoholic stimulants. While there is no

doubt that the subjects of inherited gouty tendencies are better without any form of liquor, this is frequently not so in the case of the patients more or less habituated to the use of stimulants. In judging of the suitability of the various liquors we must have regard to the usual methods of preparation of the individual beverages, *e.g.* beer, claret, champagne, etc., and to their common defects as recognised by experts in the trade. While these are outside the scope of this work, I think it well to indicate that their appreciation helps to explain the diverse views often expressed regarding the various liquors.

The free consumption of *water* can be safely recommended to many, but not to all, gouty subjects. In the case of stout adult plethoric subjects it may be advisable to restrict its use to early morning and late evening indulgence. The water is, as a rule, best taken on an empty stomach. The use of potash and lithia waters and various table waters are elsewhere referred to. With regard to *tea*, *coffee*, and *cocoa*, when suitably prepared these beverages may be partaken in moderate amount, but idiosyncrasies in their use are very often encountered. To some, cocoa is specially injurious, to others the daily consumption of coffee is soon followed by digestive and other disturbances, and in these cases special restrictions are called for. The amount of sugar allowed should be small. All *sweet beverages* should be restricted or cut off, more especially if they be in addition aerated. With regard to *alcoholic beverages*, points already referred to are of supreme importance. The decision as to what any given case can take, may only be arrived at after a careful study of the history, diet, and state of muscular activity in each case. There is no doubt that malt liquors and sweet wines are much more injurious than other liquors.

The sweet wines include champagne, Maderia, port, sherry, Malmsey, and Tokay; also porter, ale, and cider. Burgundy, Bordeaux, Rhine, and Moselle are almost void of sugar, and are therefore more suitable. The greater acidity of Burgundy and the Rhenish wines makes them, on the whole, less suitable than claret and Moselle wine.

German beer, *e.g.* lager, can frequently be taken with impunity, when even one glass of the British beers will induce some acute disturbance.

Medicinal Treatment and Mineral Waters

Some idea of the relative importance of medicinal remedies will have been gained from what has been already said in the general introduction to "Therapeutics." What we know with certainty may be summed up in the statement that all drugs which exert a normal diuretic action have a beneficial influence, and the greater the diuresis the more the benefit. Opinions will probably always vary as to the respective values of salts of potassium, lithium, and sodium, piperazin, urotropin, colchicum, salicylate of soda, guiacum, and other remedies, and individual experience will lead to perfectly honest but very divergent opinions regarding them. The more powerful diuretic action of citrate of lithium and citrate and acetate of potassium makes them occupy a relatively higher position than the various soda salts. Luff has recently expressed the view, based on experimental observations, that the salts of sodium are to be deprecated owing to an influence they exert in accelerating the transformations of the gelatinous biurate to the crystalline form. Sir William Roberts held a similar opinion. General clinical experience does not corroborate this view, *e.g.* it is not reconcilable with the benefit undoubtedly derived from the copious internal use of the Wiesbaden "Kochbrünnen," which may be regarded as a strong solution of sodium chloride. Nor is it supported by facts observed nearer home by many who use the sodium salts freely in combination with potassium or other salts, and it is perhaps wise to refer the greater value of potassium salts to their more powerful diuretic influences. An occasional short course of mineral waters is also an admirable measure. With regard to salicylate of soda my experience is in harmony with those who have not found the drug of any service in cases of typical gout. Of its value in some cases of chronic rheumatism, either when taken alone or in combination with quinine or other tonic, there can be no reasonable doubt, and it is advisable to give it a trial in doubtful cases. A useful mixture in some cases is Sodii salicylas, lithii salicylas, potassii citrat. āā gr v. t. id. Various made-up combinations with colchicum lately in the market may also be tried, but much need not be expected from them.

Salol (5 to 10 gr. t. id. on an empty stomach) and salicylate of bismuth (20 to 40 gr.) may also be useful in cases with subacute gastro-intestinal disturbance, but these must only be looked upon as adjuncts to appropriate treatment, and their use can only be necessary as the result of faulty dietary, usually combined with imperfect evacuation of the bowels. Guiacum has been well spoken of, but its taste seriously interferes with its recommendation. The special value that attaches to the use of sulphurated waters leads to the belief that the continual administration of small doses of sulphur might be attended with marked benefit; and I have lately seen a patient, a shrewd man of affairs, with pronounced uratic deposits in the palms of both hands, and an unusually extensive knowledge of health resorts both in this country and abroad, who assured me that he had of late derived great benefit from the use of this remedy in the real old-fashioned way—a bag of sulphur in his trousers pocket.

He assured me that marked relief of the pains had followed this treatment, and that no benefit had accrued until his scarf-pin showed marked discoloration. Such stories have to be received with caution; but we know so little about the rôle of sulphur in the animal economy, that no harm will accrue from our keeping our minds open to new suggestions, especially if these prove to have any basis in clinical experience.[1] In the meantime we can sum up by emphasising the fact that the medicinal treatment of gout is by far the least important part of treatment and at the same time we must indicate two dangers attendant on their use. In the first place, undue stress is often laid by the patient, more especially in the earlier stages of the disease, on the drugs in use, to the detriment of the more important dietetic regime necessary; and, in the second place, the excessive use of alkalies, mineral water, salicylate of soda, etc., occasionally does much harm by the general depression attendant on their abuse.

In the selection of a general tonic for gouty subjects, iron should as a rule be avoided, as it very often disagrees.

Mineral Waters

The various waters in common use are recommended for one of the following reasons:—

i. A purely purgative action.
ii. A diuretic action.
iii. A diuretic and medicinal action.

Of the Purgative Waters.—Franz Joseph is one of the most palatable, and its action is mild. Apenta, a bitter and faintly sulphurated water, is also mild and much favoured. Hunyadi Janos, Æsculap, Friedrichshall, and Carlsbad are all bitter aperients, with a more powerful action, but are all suitable for occasional use. The action of these waters is hastened by admixture with hot water, and they should be taken the first thing in the morning. The continual daily use of any mineral water or saline purgative is not, however, desirable.

Of the Diuretic Waters.—Some are palatable, and are taken as table waters; others are specially suited for a short course of home treatment, and should be taken thrice daily on an empty stomach for a period of two or three weeks. The table waters, which are aerated, include Apollinaris, Brieresborn, Johannis, Seltzer, St. Galmier, and Salutaris, which is a distilled water of English manufacture. The other group is represented by Contrexéville, a mild water containing sulphate of lime and magnesia, and Vichy water (bicarbonate of soda the main saline), of which there are several varieties. Célestins is the best known, but the Grand-Grille and Hauterive may also be employed with benefit. Sulis water from the springs at Bath is also useful.

Diuretic and Medicinal Action.—Occasionally the presence of anæmia, rheumatism, or some pelvic disorder suggests the use of other waters. Thus Levico (arsenic and iron) is useful in anæmia. So also is Schwalbach (Weissenbrunn preferred) and La Bourbule, the former being especially useful in rheumatic cases. Kissingen, a saline gaseous aperient water, is valuable in various uterine and other pelvic disorders in gouty subjects.

The foregoing only represents a few of the mineral waters available; artificial representations of many of these and other waters are prepared in this country and sold in tabloid form.

Spa Treatment

The beneficial effects undoubtedly derived from spa treatment in many instances arise from a variety of circumstances. For the time being worries and cares are lost sight of, the mind is at rest, and more or less pervaded by that feeling of hopeful expectancy that is of such paramount importance in treatment. The change of air and surroundings, the greater attention to diet, and the general feeling of *bien être* engendered by the interests and amusements which form an integral part of every well-regulated spa, all tend to favour that return to health which has as its most striking manifestation a re-establishment of a nitrogenous equilibrium. The great importance of the skin as a channel for the elimination of urea and other waste products has already been referred to. The local measures associated with a course of balneological treatment may best be indicated by illustrative cases.

The *choice of a spa* will depend on various things, the most important being the temperament of the individual and the special manifestations of the disease which the patient exhibits. It is needless to recommend patients to whom excitement and amusements are part of the breath of life to go to one of the quieter health resorts, while on the other hand there are not a few to whom a small quiet place is a much greater attraction. A very short summary can only be given here.

For Articular Gout.—The thermal waters of Bath and Buxton rightly maintain a high position, Bath being more suitable for the spring and autumn months on account of its mild climate, and Buxton more appropriate in summer owing to its more bracing air. Woodhall Spa (Lincolnshire), with its bromo-iodine waters, and the saline sulphurous chalybeate waters of Llandrindod (Radnorshire) can also be highly commended. The brine baths of Droitwich (Worcestershire) are of peculiar value in re-

[1] It is quite possible that this old-fashioned and apparently unscientific treatment may not be so unscientific as is imagined, *e.g.* compare the most recent method of treatment of syphilis based on the fact that the mercury is absorbed by volatilisation, and not at all by inunction.

ducing the stiffenings and deformity arising from recurrent articular attacks. The Droitwich waters are only available for external use. Aix-les-Bains, Aix-la-Chapelle, and Schlangenbad occupy a deservedly prominent place in the list of foreign spas.

Chronic and Irregular Gout.—In addition to those already mentioned, we have in this country Cheltenham, Lamington, Harrogate, and Strathpeffer. The sulphurous waters of Harrogate, Strathpeffer, and Llandrindod, are especially useful in gouty affections of the skin and mucous membranes. Lamington is well spoken of for cases of gouty glycosuria. The various foreign spas in virtue of their varying chemical composition lend themselves more readily to further differentiation. Thus we find Vichy, Homburg, Kissingen, Vals, and Wiesbaden especially useful in cases of chronic gastric and intestinal derangements. In states of plethora with hepatic torpidity, Carlsbad and Marienbad are more appropriate, and for urinary disorders Contrexéville, Neuenahr, and Vichy, deservedly occupy a high place. The sulphur waters of Aix-la-Chapelle, Aix-les-Bains, Baden (Switzerland), and Soden (foot of Taunus mountains), are good for cutaneous disorders and catarrhal states of the various mucous membranes. Soden is a quiet, little, attractive place practically unknown to English visitors, but its numerous springs compare most favourably with those of much better-known places. Schwalbach and Pyrmont are two other very attractive health resorts, and are especially good for anæmic, debilitated, gouty subjects. In view of the great value of medical gymnastics, especially when combined with balneological treatment, it may be well to mention the leading spas which have a fully equipped mechanico-therapeutic institute. These are Aix-la-Chapelle, Baden-Baden, Wiesbaden, Wildbad. It is unfortunate that in this country, where gout has, as it were, its headquarters, more attention is not paid in our medical schools to instruction in dietetics and in mechanico-therapy — the two fundamentally important factors in treatment.

Acute Gout—Recent Attacks

These cases are the most suitable for the attainment of an excellent result, and the more recent the attacks the more hope there is of a speedy and permanent beneficial result. Needless to say the details of treatment will vary, depending on the constitution of the patient and the nature and strength of the waters employed. The course should last not less than five or six weeks, and its daily routine should be along the following lines: — *Diet.* — The dietetic treatment should be conducted along lines already laid down, the food being simple, and composed mainly of light animal foods, eggs, vegetables, and the more easily digested bread stuffs. Preferably no alcohol or wines should be taken, but if that is not feasible, whisky and potash, or lithia water, or a little sound claret are the most suitable. On no account should liquors be mixed. *Exercise.*—Ample exercise should be taken, a course of passive movements supplementing the patient's own exertion in the matter. There is much more danger in delay in moving the affected joint than there is in unduly exercising it at an early stage. In the case of the upper extremity there is greater necessity for a special course of passive movements. *Baths.*—If patient is a robust subject, he should take a curative bath daily, but in weakly or nervous subjects every other day will suffice, at any rate at the outset. A feeling of slight excitement, a sense of general discomfort, loss of appetite, or sleeplessness, are indications pointing to the advisability of prolonging the intermissions, or it may be, diminishing the temperature and strength of the bath. During this treatment it is not an uncommon experience for acute pains to develop in the affected joints, even a further acute attack may supervene. Such occurrences are usually ascribed to the searching nature of the remedy, and if not immoderate need not be regarded with disfavour. The best time for the bath is the early morning, an hour to an hour and a half before breakfast, and it should be taken on an empty stomach. (To those patients who cannot or will not bathe fasting a cup of coffee or tea may be allowed.) The temperature of bath ranges from 80° to 105° F., and depends on the susceptibilities of the patient. He should remain up to the neck in water, and continue to exercise the affected joints by active movements, and by friction and kneading of the parts. The duration of the bath should be from eight to ten minutes to start with, and be gradually increased up to twenty or thirty minutes. The duration, however, largely depends on the temperature of the bath. After drying and using moderate friction with a roughish towel patient should lie down for half or three-quarters of an hour, and thereafter dress himself in clothing appropriate to the time of year. He will then be ready for his breakfast. The warmer baths should only be used for stronger and more vigorous subjects, as they are liable to upset more weakly or nervous ones. *Drinking Cure.*—This consists in drinking the waters in an amount varying from one and two pints thrice daily on an empty stomach, *e.g.* 8 A.M., between 11.30 and 12.30, and between 4 and 5 P.M. As a rule no further measures are called for, but the use of the douches and various forms of local hot-air or other applications are useful in appropriate cases.

A Case of Chronic Gout with Irregular Manifestations

Here the regulations already laid down for diet and exercise should be even more strin-

gently enforced. The latter measure especially is only too frequently not given adequate effect to in spa treatment. Carbohydrates and sweets must be reduced, or even abolished, and great care taken to avoid admixture of fruits and carbohydrates with the chief proteid foods. A large quantity of water should be drunk in the course of the cure, beginning, however, with small amounts ($\frac{1}{2}$ to $\frac{3}{4}$ pint thrice daily), and every third or fourth day gradually increasing up to two or even more pints thrice daily. The amount should be carefully regulated, and is largely dependent on the cardio-vascular tone of the individual. Special care should be taken with stout subjects, who are also less liable to take the amount of exercise requisite daily. The baths should only be taken every alternate day, or in some cases less frequently, but in the intervals various accessory local measures are of the greatest service. These comprise the douche (*vide* vol. i. p. 347), active and passive movements of various kinds, which are most readily obtained by a course of exercises in a well-equipped Swedish mechanical institute, and various forms of local hot-air treatment. A very good general rule to adopt with hydropathic and allied remedies intended to influence local metabolism is to use them "little and often."

Hydro-therapeutic measures are not as a rule applicable in cases of gouty cachexia, nor in elderly persons especially with obese tendencies, and further, their use requires special consideration in cases where any organic, cardiac, renal, or pulmonary lesion exists. The degree of general limpness and enervation so frequently experienced in the earlier part of the course reflects the profound alterations that are taking place in general metabolism, and these may be still further indicated by the occurrence of a marked oxaluria or phosphaturia. After a course of spa treatment it is often desirable to endeavour to thoroughly establish the cure by sending the patient to some alpine or other health resort for a time, where the climate is thoroughly invigorating and calculated to promote general tone and vigour to the system.

Treatment of Acute Gout

Acute gout falls to be treated on the general principles applicable to other inflammatory states. The indications are to relieve the constitution, and to moderate the local inflammation, both of which are best fulfilled by constitutional rather than local treatment. Rest in bed, free purgation, and a low diet, are the essential constitutional remedies, the two latter being employed in proportion to the acuteness of the symptoms and the constitutional vitality of the individual. In weakly and in old subjects, and in cases where the gouty cachexia is pronounced, the treatment must be less depressant, and a more generous diet with some alcoholic stimulant may be indicated. On the first suggestion of the paroxysm in an otherwise healthy subject, a full dose of calomel (4 to 8 grs.) and colocynth (2 to 4 grs.) should be taken and followed by a saline cathartic (Carlsbad salts). In a few cases this treatment suffices to arrest a paroxysm, but if the attack is fully developed its remedial influence is not so noteworthy. Recourse should then be had to active diuretic treatment and the administration of colchicum. Large doses of lithia or potash water should be taken at least three times a day, and in addition fifteen to thirty minims of the vinum or tincture colchici should be given in a little water, or in the following mixture thrice daily:—Vin. colchici ℥ss., pot. citrat. ℥ss., lithii salicylas ʒj., aqua ℥vj.; ℥ss. in water thrice daily. Great care must always be exercised in the use of colchicum, as not a few subjects react strongly to its influence, severe general depression or diarrhœa resulting from even moderate doses. How the drug acts is unknown. There are no conclusive indications that it influences the excretion of uric acid. The very slight increase noted by His in the most recent observations on the subject are not (*Deutsch. Archiv für klinische Medicin*, Oct. 1899) distinctive, and we may confidently assert that the beneficial effects of the drug are not dependent on any direct influence on uric acid. Sir Dyce Duckworth has suggested that the result may be explained by the cholagogue action of the remedy. Salicylate of soda is another remedy that has been found useful in a few instances of acute gout when colchicum was unavailing. In the few cases that I have tried it in acute gout I have never been satisfied that it exerted any therapeutic influence. Ten to fifteen grs. of Dover's powder, or a combination of bromide of lithia (30 grs.) and chloral hydat. (30 grs.) may be indicated for their sedative and hypnotic action. This treatment must be carefully revised from day to day in the light of the patient's general state, and notably the condition of the pulse, which may be taken as a reliable guide as to whether the depletent remedies are being administered in excessive amount. The diet should be limited to milk only for the first few days, or milk alternately with weak beef tea. Solid food of any kind is better withheld in robust subjects—nothing but benefit will accrue from a mild starvation diet. In a few days bland farinaceous food may be added, provided the state of the tongue is satisfactory, and all flatulence and eructations gone. Gradually, white fish, boiled chicken, eggs, and easily digested bread stuffs, are added to the diet. With regard to local treatment, elevation of the limb, a covering of wool surrounded by oil silk, a bandage, and a cradle to protect the limb from the weight of bed-clothes, are usually all that is required. Of other measures the most appropriate are flannel fomentations wrung out of hot water and sprinkled freely with laudanum,

or the application of lint soaked in lin. belladonna and tinct. opii. When these are not available, lint soaked in methylated spirit or one of the cheaper whiskies makes a fair substitute. All cold applications, blisters, leeches, and the like should be avoided. Dr. G. Balfour has found the application of steady manual pressure to the joint from the outset of symptoms to be attended with good results. Such drastic measures will probably, for obvious reasons, have only a very limited application. As soon as all acute manifestations are gone, active and passive movements of the joint should be begun steadily and persevered in. In subacute attacks the various troublesome local complications met with, *e.g.* thrombosis, œdema, eczema, etc., must be treated on general medical principles, but their too zealous treatment is to be deprecated.

Illustrations of Treatment

From the foregoing outline of treatment it will be evident that the therapeutic measures vary enormously with the different stages and very diverse manifestations of the disease. As previously stated, every case of gout is a new problem in therapeutics. I have thought it well to give further point to this by recording in detail a series of illustrative cases, and have selected a few which have been under my observation for a lengthened interval, so that I am in a position to speak with accuracy of the effects of the different regime recommended in each case.

1. Mr. A., 16, schoolboy.—Marked hereditary history of gout.

   i. Erythematous eruptions.
   ii. Erythema elevatum diutinum.
   iii. Pharyngitis and tonsillitis.
   iv. Nail disorders.

This case is selected to illustrate some clinical features of gout in the young subject and also to emphasise the rational prophylactic treatment of the disease. The patient is a tall, big-boned, muscular subject whose weight is distinctly above the average for his height and years. He excels in athletics, being an adept football player, golfer, and swimmer. His home surroundings have been of the kind most favourable to the full indulgence of a keen appetite and sound digestion. His diet is plentiful and rich, and all his life he has been in the habit of drinking large quantities of milk independently of much nitrogenous food at ordinary meal times. Scotch oat cakes, butter, and jam are a special weakness. He is a total abstainer.

About the age of 8 or 10 he began to show a tendency to the frequent development of "skin spots," whose onset seemed to coincide with a slight excess in diet. From the descriptions available there seems to have been discrete erythematous patches, but, whatever their nature, they soon disappeared under the influence of a purgative and restricted diet. Three years ago he was the subject of a pronounced attack of erythema, affecting especially the fingers, back of wrist, and to a less extent the knees and upper part of face. These parts were swollen, red, burning hot, and extremely itchy. The patches on the back of the wrist were markedly raised and corresponded closely to the conditions described by Crocker, "erythema elevatum diutinum," as occurring in gouty subjects about the age of puberty. The conditions disappeared under the simple treatment above mentioned. Since that time patient has had no similar attack, probably because, on the slightest appearance of it, which is by no means infrequent, the necessary therapeutic measures are taken. He has found by experience that if a saline be taken sufficiently early the other remedial measure is not necessary, and he acts accordingly. The reliability of his own observations and the extent of his faith may be gauged from the fact that he seldom goes off for a holiday without a supply of his favourite mineral water. On more than one occasion when he has done so, he has felt seriously out of sorts within a very few days, the most striking *objective* symptoms being the development of swelling, redness, and pain in one or more fingers. I have not seen this condition; but his mother, who has an accurate lay knowledge of the disease, affirms that it is "either gout or chilblains." She recognises no relationship between the two, and, when her inclinations lead her to write for a remedy for chilblains, all her knowledge of facts prompts her to adopt the remedies appropriate to gout. The patient is also subject to occasional attacks of inflammation of the pharynx and tonsils, the inflammation lasting for 24 or 48 hours, and resembling more the appearance of a general swelling and acute congestion than a bacterial invasion. A year ago he had some trouble with his nails. A transverse furrow developed at the roots of two of his toe nails and one finger nail, and gradually the nails were cast off and replaced as after a traumatism.

With regard to the diagnosis of gout in this case, while some may question its accuracy, I am perfectly satisfied that the manifestations of disease described are directly due to the patient's gouty proclivities. It is a case of gout in the young and robust subject.

The case admirably illustrates the treatment appropriate to the disease. It is clear that his symptoms tend to develop after a slight or marked excess in diet, more especially if jams or other sweets have been largely consumed, on which occasions there have been no abatement in the amount of red meats and other nourishing foods. The saline, which may sometimes be associated advantageously with blue pill, acts beneficially in two or three ways. It removes from the alimentary canal certain toxic substances produced locally as a result of

defective metabolism, which act directly as a local irritant, and by their removal as well as by the withdrawal of any other indigested food-stuffs in the alimentary canal a condition of rest is established in the general cell metabolism, which enables the tissues to cope more successfully with the surfeit of nutritious matter to which they have been subjected. This latter is rendered more easy, if, as is generally the case, the patient's diet for the few following days is a more strictly physiological one. The depletion effected by the rapid withdrawal of some fluid by the alimentary canal doubtless assists the re-establishment of a general equilibrium. The occasions of these various congestive attacks in a young subject are probably, as with the paroxysms in adults, to be explained as the result of long-standing over-nutrition, with deficient elimination, and the excess immediately preceding the attack has merely strained the capacity of the tissues—intestinal viscus and general cell life—to breaking point. The general plan of treatment appropriate to such a case may be briefly summarised—

i. Moderation in diet—a slight all round diminution of the total quantity of food consumed at each meal; the acquirement of self-control.

ii. Restriction and gradual cessation of the milk-drinking between foods, and also a diminution in the amount of fluid drunk at meal times. His habit is to drink very large quantities of fluid at the different meals, which fluid would be more wisely taken in the intervals.

iii. Limitation of the jams, sweets, and fruits, of which he partakes inordinately, also care in the consumption of the various sweet summer beverages. (These patients are as a rule addicted to sweets of different kinds).

iv. Daily satisfactory evacuation of the bowels, by artificial means if necessary. In any case, to be supplemented by an occasional saline (once in 8 or 10 days).

No other restrictions are called for. Nor are any recommendations as to exercise, hydropathic or other measures, the least necessary so long as the patient's inner respiratory activity is at the high level entailed by his being in first rate physical training. However, it is well again to refer to the fact that the patient finds that strict attention to the last of these directions alone suffices to keep him free from any active manifestations of his gouty tendency, and he acts accordingly. This circumstance in no way invalidates the importance of the other recommendations, the neglect of which will certainly, in course of time, with the altered habits incidental to adult life, lead to other characteristic developments of a minor or major type. Succeeding years of indiscretion progressively increase the hold of the disease on the tissues, and further experience of the case may illustrate that the efforts of nature, even when aided by a much more strict regime than that just recommended, are quite incapable of extinguishing the disease.

2. Mr. B., æt. 26, mason.
Chronic articular gout.
Lithæmia.
Acute exacerbations.

This case is selected to illustrate some points in the dietetic treatment of the disease in its more acute phases. The patient was a hospital one, and a full record of his case with a series of laboratory observations made on his general metabolism are in course of publication (*Journal of Pathology*, summer 1900). I have not the same knowledge of the later history in this case as of the others; but the points that I wish to emphasise are sufficiently brought out by the notes available. Unlike the others recorded, this patient was in the habit of passing large quantities of free uric acid, and he was thin, weak, and slightly cachectic in appearance. The salient features are as follows. The extent of his weakness may be gauged from the fact that two months after the acute attack the record of the dynamometer gave—R. hand, 55; L. hand, 35:—

The hereditary history showed the father to have been subject to rheumatic fever, the grandfather to have been rheumatic, and one brother to be subject to "attacks like the patient." He enjoyed good health until fourteen years of age, when he had a "rheumatic" attack lasting three weeks. When æt. twenty, similar although less severe attacks developed at intervals, and when æt. twenty-three a specially severe one lasted thirteen weeks. When æt. twenty-six, patient was admitted to hospital suffering from well-marked acute polyarticular manifestations, supervening in joints and tissues already the seat of chronic deforming gout. The ankles, knees, hands, elbows, and ears were all involved, crystals of urate of soda being readily obtained from the deposits in the ears. Patient had been a fairly heavy meat eater, a temperate drinker, addicted to very little exercise, and accustomed to the exposure necessitated by his trade, which entailed a considerable amount of travelling from place to place. He did not think that diet had played an important part in the manifestation of symptoms, but he attributed an influence to the drinking water of various localities. Tomatoes were a special weakness, and he had partaken freely without apparent detriment.

After recovery from the acute attack patient was gradually put on a light hospital diet, as follows:—

Breakfast.—Porridge and milk, toast, tea, and frequently an egg.

Dinner.—Soup, bread, and fowl, fish, or red meat, with potatoes; or fish, fowl, or meat

and potatoes, with a pudding; occasionally green vegetables.

Tea.—Tea, bread and butter.

And in addition a cupful of milk was taken either alone or with soda water twice daily. During this time patient was making a very slow recovery, being continuously very subject to sharp recurring pains in different regions, excessive weakness in the feet, hands, etc., and he was in this state when he left the hospital to go to the Convalescent Home for four weeks.

His diet then was as follows:—

1. Porridge, tea, bread and butter.
2. Fish or fowl, bread, potatoes, rice or other milk pudding.
3. Tea, bread and butter.
4. Porridge and milk.

The most important differences between this and his former diet were the absence of all soups, red meats, and green vegetables. Under this regime he improved very materially, and, for him, very rapidly. The pains diminished and soon disappeared; he gained $6\frac{1}{2}$ lbs. in weight in four weeks. He now felt well.

On his return to hospital for further observation his diet was inadvertently changed back to his former hospital diet with immediate unfortunate results. The pains reappeared, the stiffness and weakness became accentuated, and at least one of the former weak spots became swollen, red, and tender within thirty-six hours after his return to hospital. His appetite and digestion remained apparently unaffected, the tongue was slightly furred, and the total amount of food consumed was actually less in amount than it had been at the Convalescent Home. No actual acute attack developed, but his whole metabolism was deranged and he lost $3\frac{1}{4}$ lbs. within six days. Even allowing for the change of air incurred on his return to the hospital, the case was a perfect picture of the profound influence of diet on the disease. If we analyse the altered circumstances which led to the derangement of metabolism, there is no doubt that the soup was prejudicial, and mainly because it interfered with the normal local metabolism in the proteids in the diet. There is also every reason to believe that the milk taken in the course of the hospital diet was not calculated to give the various glandular secretions the rest requisite for their due functional activity. Other points there may be, but these will suffice. In gout, as in health, the tissues can cope with a range of diet within which no untoward effects result. The "convalescent" diet was the more physiological one, and a reference to its nature and amount clearly shows what nature is willing and able to do. Further, that diet could certainly be modified in different directions, *e.g.* to include vegetables without detriment so long as the necessary rearrangement was made.

There are not a few cases like this one in respect of the noxious influence of soup, but in this connection regard should be paid to the points in the composition of soup already referred to. With regard to the meat, this patient is an illustration of a type in which all red meats are better avoided. Whatever the exact cause may be, there are cases of gout when the reaction of the tissues to intestinal influences is more marked and more unfavourable if red meats are a component of the diet, and these should be dieted accordingly. But such cases are exceptional.

3. Mrs. C., æt. 32, married; 2 children.—Hereditary history of gout.

i. Neuralgia.
ii. Pains in great toe joints and legs.
iii. Acute gout.

This patient is a big, well-built woman of active bodily and mental habits, with keen appetite and excellent digestion, which she is in a position to gratify, although she does not eat immoderately. Her weight well exceeds the average for her height and years. She occasionally has a glass of claret to dinner, and takes very exceptionally a glass of beer to lunch. The latter is a beverage of which she is particularly fond, but she indulges very occasionally, as she has long recognised that it was prejudicial to her. This case illustrates well the ill-declared type of gout that is more frequently seen in Scotland than the truly typical form.

As a girl she was subject to facial neuralgia, which, however, has quite disappeared. Since the age of 17 she has been occasionally the subject of sharp pains in the calves of the legs, and eighteen months ago she was confined to bed for two days with severe pains in the upper arm and shoulder, a condition which I was satisfied was a slight inflammatory attack involving the brachial plexus. For many years she has had occasional sharp twinges of pain in both great toe joints, lasting from five minutes to half an hour, often associated with a stinging pain in the calf of the leg and cramp-like feelings in the soles of the feet. She has also been a martyr to chilblains. Six months ago a typical mild attack of acute gout developed in the left great toe joint, which was recovered from in less than three days. The attack developed at 10 P.M. and only lasted some five or six hours, and when I saw her at the height of the pain the affected toe joint was slightly swollen, the superficial veins prominent, the skin showing a localised red area the size of a shilling, and the joint very painful on pressure. On this day she had partaken of a larger lunch than usual, and with it a bottle of beer, and to this indiscretion she attributed the attack, although she has occasionally taken beer without any accession of symptoms. That

beverage is a more active poison to her than to many others of apparently equal gouty proclivity.

The diagnosis is quite assured in this case, and the earlier manifestations of neuralgic pains, etc., may be compared with case 1, where the earlier symptoms were more vascular in origin. It has long been recognised that a proneness to inflammatory conditions of the peripheral nerves, and to a weakness in the vascular system (which may, of course, be partly of nervous origin) are characteristic of inherited gout. This relationship has been emphasised by Hutchinson. Care must, of course, be taken in these nervous cases to differentiate rheumatism, and while this may be difficult and often impossible, in not a few cases an honest and accurate diagnosis of gout may be arrived at.

What is the appropriate treatment? We have here an adult woman in comfortable circumstances, living a town life, with a husband and two children to look after, accustomed to good living, and not in the habit of always taking exercise proportionate to the full diet enjoyed.

In view of these special circumstances we must not be surprised if good results are less readily obtained, nor will it be surprising if more attention requires to be devoted to promoting the functions of the excretory organs and getting rid of the accumulated products of deficient combustion in the body. These can be judiciously influenced by exercise as illustrated by the following:—Some years ago a friend of the writer, a typical example of plethoric gout, sought advice from her consulting physician for various acute gouty manifestations to which she was a victim. She was recommended a very strict diet, and one which could not easily be given effect to even in a large, well-regulated household; she declined the recommendations, stating that she would rather die, a statement which those who knew her appetite and mode of living could quite believe. She then discussed the matter with an intimate lay friend, who suggested that she should not over-eat, and should buy a bicycle and ride it. She accepted the recommendations, regarding the former as in the light of a compromise with the advice of her medical adviser, and from that time until now she has been remarkably, although not entirely, free from all active manifestations.

The special treatment may then be summarised as follows:—

(i.) A slight all round restriction in the quantity of food consumed. The lunch to be mainly if not always a vegetable one, and red meats to be replaced by chicken or fish for dinner, at least one day in the week. Sweets of all kinds only to be partaken of sparingly. The principal meal to be taken dry, one small glass of claret to be taken at dinner if desired. (This is the only wine for which she has a liking.) Beer is better avoided in this case. Water to be drunk freely between meals, and a tumbler of hot water taken at night.

(ii.) More systematic exercise. Sharp walking or cycling in place of driving, supplemented by ten minutes' physical exercise daily, preferably after morning bath.

(iii.) Attention to the bowels. As the bowels move daily without an aperient, all that is required is the use of a mild yet active saline every week or ten days. Further measures will probably be necessary at the monthly periods, as the patient, like many others of a like habit of body, is prone to undue states of depression in the premenstrual period.

(iv.) Hydropathic Measures. If the foregoing measures are duly carried out, the only hydropathic measures called for will be fulfilled in the daily morning bath followed by active friction, supplemented by a hot bath once a week. But as a matter of experience, the first three measures are only imperfectly carried out, with the result that more active eliminative treatment becomes a necessity. The choice of a spa is in the main immaterial so long as the important elements of cure are secured. These are, a complete change of air and surroundings in a climate where temperature and other atmospheric influences will conduce to a reasonable amount of muscular activity, a simple diet, judiciously flushing out the system by a quantity of one to three pints or more fluid daily at intervals on an empty stomach, and a course of baths two to three times a week, adapted to promote vigour and tone in the skin. Special care has to be exercised in the amount of water to be drunk, and the number and form of the bath to be recommended in stout flabby subjects. While this patient is not flabby, she reacts unfavourably to even a mild Turkish bath, and has also always reacted badly to cold sea bathing.

4. Mr. D.,[1] 38, tailor.
Chronic tophaceous gout.
Surgical interference.

I have selected this case because it illustrates so well some other points in the management of gouty subjects. It is additionally interesting owing to its severity, such a case being almost unique in a Scottish hospital. My attention was first drawn to it by Mr. Alexis Thomson, under whose surgical care he was, and to him I am indebted for the record. During the past year I have taken an opportunity to see the patient frequently, and obtain from time to time an accurate record of the results of treatment. The successful results derived from the limited therapeutic measures employed clearly indicate than any line of treatment found

[1] The surgical aspects of this case are recorded in the *Edinburgh Hospital Reports*, vol. vi. "Gouty Formations in Tendon Sheaths, Bursae and Skin (with Photographs)." By Alexis Thomson.

beneficial in a given case of chronic gout should be considered applicable only to the individual in question, and no generalisations whatever should be made from it applicable to the disease. There is no single line of attack on the disease. Every case of gout has to be considered on its own merits.

The patient is a small, thin, rather pale, but not cachectic subject. There is no hereditary history of any importance, but the patient knows very little about his family. As a young man he was a keen football player, and partook freely of port wine and whisky. His diet has been the ordinary mixed diet of his class, the only noteworthy point being his strong aversion to fats. His occupation conduces to a very sedentary life. About the age of 28, shortly after the end of his football career, he had his first experience of acute gout, which involved the ankles. About eight or ten months later he had a second attack, involving the wrists and back of hands, and thereafter he had an attack about three times a year, each one incapacitating him for about two or three weeks. During these attacks he was kept on a light febrile diet, but in the intervals his food was an ordinary mixed diet, with occasional indulgences in liquor. Two years ago he was off work for seventeen out of the fifty-two weeks of the year, this period representing at least three severe attacks. At this time, even in the intervals, he was much troubled with pains and discomfort in connection with the numerous gouty deposits that were now developed, the position and size of the swellings interfering with the sitting position adopted by tailors, and also with his sewing powers. About eighteen months ago his medical adviser suggested surgical interference, and he was admitted to hospital for this purpose. He then presented all the typical features of a very pronounced case of chronic tophaceous gout. The ears were the seat of numerous characteristic gouty deposits. The extensor aspects of several fingers, especially those of the right hand, most used at his work, showed the presence of nodular swellings, varying in size from a small bean to a small marble. It was not easy to determine whether these were truly subcutaneous or connected with the tendon sheaths. The second joint of the right middle finger was enlarged, swollen, and tender. The subcutaneous tissue of the feet and ankles was the seat of several large swellings, varying in size from a marble to that of a small orange, one of which had burst and exuded crystals of urate of soda. A similar swelling was present over the left olecranon process. The joint structures as a whole were remarkably free. Five of these tumours were removed, two from the neighbourhood of each ankle, and one from the elbow. They consisted of masses of urate of soda crystals, embedded in thickened and degenerated connective tissue.

Patient has enjoyed very much better health since the operation, and he has not been a day off work for more than a year. His regular diet has not been materially changed, but both he and his wife agree that he is now almost a total abstainer. His diet is now as follows—

Breakfast.—Porridge and milk. One and a half breakfast cups of tea, with moderate amount of sugar. Bread and butter, and usually an egg or fish.

Dinner.—This may be either soup (broth, rice, or potato) and meat (boiled, stewed, or steak), or meat and a pudding (suet puddings of all kinds and rice puddings). Green vegetables are taken probably two days a week; potatoes are taken sparingly, and bread in fair amount.

Tea.—An egg or fish; bread, toast; one and a half breakfast cups of tea, and frequently jam.

Supper.—Glass of milk, often with a little bread and butter.

In addition to the foregoing he drinks large quantities of water between his meals, and under this regime he now enjoys practically perfect health, although the right middle finger may occasionally give him a little discomfort, otherwise the tophi originally present have remained unaltered. No hydrotherapeutic or other special remedial measures have been employed, for obvious reasons, and yet the results of treatment must be regarded as highly satisfactory. Such a result must, however, on no account be interpreted as minimising the great value of hydrotherapeutic and other measures calculated to stimulate the eliminative powers of nature and promote a more healthy tone and general metabolism. It is probably more wisely interpreted as indicating that when nature is relieved from the injurious effects of even one noxious agent (in this case the alcohol), she may find herself competent to restore a fair nitrogenous equilibrium on a rational mixed diet. In this case surgical measures certainly assisted to give him a fresh start in the path of normal metabolism.

5. Mrs. E., 55.
   - i. Peripheral neuritis and vascular disturbances.
   - ii. Lumbago.
   - iii. Headaches.
   - iv. Tophi.

Mrs. E. is a stout, typically gouty-looking subject, of plethoric habit, who has lived an active town life and has lived well. Of late years she has restricted her diet slightly, more, however, in accord with the teachings of her own experience, than as a result of medical advice which she seeks, but does not readily follow. There is a distinct hereditary history, a brother and two uncles being afflicted with well-defined gouty manifestations. Patient has

on the whole been a very healthy woman; she has had ten children.

When thirty years old, and after the birth of her second child, she was the subject of periodic attacks of very severe pain in the sole of one foot. This pain would last from two to four hours, and completely incapacitated her. During the paroxysms the foot looked bloodless, and felt cold, and was usually treated by being placed in very hot water, followed by elevation of the foot above the rest of the body. The onset of these attacks could not be accounted for, but occasionally one developed after over-fatigue. Pains of a somewhat similar, although much less severe nature, are occasionally complained of now, but patient believes that these are prevented, or very much relieved, by the continuous wearing of a tight foot bandage, which she has worn for more than twenty years. The conditions resembled that now described as metatarsalgia (see vol. ii. p. 292). She has had several severe attacks of lumbago, which have usually occurred towards the end of the summer months, and have been associated in the mind of the patient as the penalty of indulgence in fruits of various kinds. This association has been present sufficiently often to attract her own attention, but it has not influenced her habits very much, and for the last five years she has quite expected the annual visitation, which, however, has been of a mild character. Her vaso-motor system has long been in an unstable condition. When fatigued, or more readily on dining, especially if the meal commence with a hot soup, a red flush (one or more) develops on the cheek, nose, or chin, and remains for some time. Of late years she has been subject to very severe headaches, and about a year ago, patient came under my observation owing to the development of a small tophus over the sixth rib on the right side, which had burst quietly, and was exuding crystals of urate of soda. An examination of urinary and cardio-vascular systems reveals no evidence of chronic kidney disease (sp. gr. 1016), but a careful consideration of her history and constitution led me not to lay much stress on the absence of external evidence of renal weakness. Her menstrual life had been characterised by frequent excesses, but never such as to call for special treatment, as she was fully aware of her plethoric habit of body. One thing that has probably saved her from a much more active manifestation of the disease is that strict attention which she has all along paid to the function of the bowels. The constant use of an appropriate aloin pill and frequent reference to salines never allowed any important derangement in this respect. Patient is one of those gouty people who value medical advice only in a theoretical way, and keeps in the wake of knowledge gained by self-instruction, but her experience has led her within recent years to give up taking soup and meat to lunch, and to lessen the amount of sweets and fruits. Various aches, pains, and tender feet are readily induced by any indiscretion, and such is by no means an infrequent occurrence.

What is the appropriate treatment? Owing to the long-standing nature of the disease, with the associated greater instability of tissue metabolism, and lessened power of resistance in the intestinal tract, the treatment will obviously differ in detail from any of those previously recorded. It may be well, in the first place, to give in full the diet which she has taken and finds well adapted to keep her free from gouty manifestations.

Breakfast.—Avoiding kidneys, steak, liver, and all made-up dishes, and only taking one solid, *e.g.* fish, of any kind (except salmon and fatty fishes which do not agree with her), one or two eggs, or bacon and egg; one breakfast cupful of freshly-made tea with not more than one piece of sugar. Toasted bread, Vienna rolls, or other bread (not too new or doughy), with butter. Marmalade or jam to be taken only in very small quantity, and even then not to be taken every day.

Lunch, to be mainly vegetables.—Selections from the following: tomatoes cooked or plain, macaroni dressed in various ways, salads, celery, cauliflower *au gratin*. Bread, brown bread, Vienna rolls, or any form of unsweetened biscuit; small piece of mild cheese, if desired. Small quantity of fruit, one of the following: an orange, raisins, apples, figs, dates, walnuts. Half an ounce of whisky in half a tumblerful of soda water.

Afternoon Tea.—One or two small cups of tea, with a very thin slice of bread and butter, or a piece of very light cake. Very little solid be taken, and especially no rich cakes.

Dinner.—The dinner to consist of three courses, to which fruit can be added, if not taken to lunch. The diet to be arranged on the following plan—

1	2	3	4	5
Soup	Fish	Soup	Fish	Soup
Meat	Meat	Entree [1]	Meat	Fish
Pudding	Savoury	Pudding	Pudding	Savoury
Fruit		Fruit		

Care has to be taken that on the nights when meat is taken the soup should be of a lighter character, *e.g.* clear brown, rice, or fish soup. Similarly on nights 2 and 4, the fish should be of the lighter kind, *e.g.* whiting, haddock, or sole. On the 5th night the soup can be richer, *e.g.* oxtail or kidney with a light fish, or the soup may be light and the fish more rich, *e.g.*

[1] Sweetbread, tripe, chickens, rabbit.

turbot, halibut, or skate. No more than two vegetables are to be allowed, and then in sparing amount—a sauce to be reckoned as a vegetable. (Half an ounce of whisky in not more than half a tumblerful of water the only safe beverage for constant use.) Claret, champagne, and whisky are the only drinks for which patient has any regard. The puddings recommended are milk puddings of various kinds, stewed fruits made with small quantity of sugar.

Suet puddings only to be taken in sparing amount, and then only when the other courses are of the lighter nature. Jellies and creams can be taken sparingly, merangues (a special weakness) to be avoided. Bananas and strawberries are best avoided. Small cup of *café noir* prepared without sugar.

Are any medicinal remedies called for? As the patient is a sensible woman and fully appreciates the very small influence which drugs have exerted in her disease, none should be recommended with the exception of the constant use of a vegetable pill, and frequent use of the saline already referred to.

This case is a fair illustration of a not uncommon type in which the purely vegetarian diet, so eloquently advocated by Haig, is not applicable in treatment, and even if it were advisable there is not the remotest possibility of its being carried out. With regard to exercise, as the patient is of an active temperament, and has a considerable amount of muscular exercise in connection with her household duties and numerous outdoor interests, no further recommendations are called for. Further, it would be well for her to arrange to have her annual holiday occasionally at a health resort, where a full course of hydropathic treatment under medical directions will be useful in working off the effects of the occasional or frequent deviations from the paths of physiological righteousness, which may be regarded as a part of the clinical history of the disease; and in the intervals, and for the same purpose, an occasional course of diuretic remedies will be found of the greatest service.

Summary.

It may be well now to tabulate the principal points which should be kept in view in the general management of all cases.

1. When a hereditary tendency exists in children, habits of extreme sobriety in eating and drinking should be cultivated, and the diet should be mainly a milk, vegetarian, and light meat one.

2. When the disease is established there is no routine treatment, and the details will not be the same in any two cases. Attention should, however, be directed in turn to the following:—

(*a*) The diet and state of the digestive tract.

(*b*) The amount and nature of the exercise indulged in.

(*c*) The functional activity of the skin and kidneys.

(*d*) The state of the central nervous system.

3. The quantity of food should be determined by the amount of active exercise. Three meals per day only to be taken, and those to be of a simple character.

4. When in doubt as to which set of food constituents to cut off, begin with the carbohydrates, and especially the saccharine substances. In not a few cases the latter may require to be completely cut off, and in every instance special care must be exercised in the admixture of carbohydrates with the fats and nitrogenous foodstuffs.

5. Thorough mastication of the food is all important, and strict attention to the evacuation of the bowels a necessity (with, in addition, an occasional saline).

6. *Fluids and Beverages.*—(*a*) Alcohol in any form is better avoided, except in subjects who are more or less habituated to its use, in which case the liquor taken should be *the one* which is known by the patient to be least detrimental to him. On no account should drinks be mixed. All sweet wines and malt liquors should be avoided, unless the patient is satisfied from careful observation that these are not prejudicial to him. The stimulant should be taken in a measured quantity and with meals.

(*b*) The free use of the alkaline table waters is to be commended, care being taken that an excessive amount of fluid is not taken with the meals.

(*c*) A tumblerful of hot water at night and an occasional course of mineral waters taken on an empty stomach are useful eliminants.

7. *Exercise.*—This should be adapted to the age and sex of the individual. Various forms of home gymnastics are useful in supplementing the course; in all cases care must be taken to avoid excessive muscular fatigue.

8. Attention to the excretory functions of the skin is of the first importance. This can be well done in the first instance at home by the daily morning bath, and the aid of a hot bath once a week or more frequently, followed by the stimulating effects of a cold spray. Flannels appropriate to the time of the year are to be worn. An occasional special course of hydrotherapeutics at a suitable health resort is desirable in all cases where such treatment is possible.

9. Medicinal remedies form the least important part of the treatment, but are beneficial in certain cases, more especially in those with a naturally weak digestive tract.

10. Owing to the profound influence of the mind upon the body, all the steps necessary to secure a state of mental rest (cessation of worry, change of air to a more bracing climate) must be advocated, and if these are not given effect to, the various other remedial measures may be of less avail.

Goux System.—A dry method for the disposal of sewage; pails with an absorbent lining (of dry peat or sawdust) are used, and this is replaced each time the pails are emptied.

Graafian Follicle. *See* Generation, Female Organs of (*Ovaries*); Fœtus and Ovum, Development of (*Graafian Follicle*).

Graefe's Sign.—Graefe noted that in exophthalmic goitre the upper eyelid does not follow the downward movement of the eyeball as it does in health; this is known as Graefe's or Von Graefe's sign. *See* Thyroid Gland, Medical (*Exophthalmic Goitre, Symptoms*).

Grafting. *See* Skin Grafting and Allied Procedures.

Gram's Method.—Differential staining with gentian violet. *See* Post-Mortem Methods (*Bacteriological Investigations*).

Granati Cortex.—Pomegranate bark is obtained from the root and stem of *Punica granatum*, and is used as an astringent, and, occasionally, as an anthelmintic in cases of tapeworm; it contains alkaloids (*pelletierine, isopelletierine*, etc.) and punico-tannic acid; and there is an official preparation, the *Decoctum*, the dose of which is ½ to 2 fl. oz.

Grancher's Disease.—Under the name of spleno-pneumonia, Grancher described in 1883 a new disease of the lungs. It occupies an intermediate position between lobar pneumonia and pulmonary congestion, and is a kind of subacute pneumonia which simulates pleurisy with a medium amount of effusion.

Grand Bouillon. *See* Invalid Feeding (*Varieties of Soup, Stock*).

Grand Mal. *See* Epilepsy (*Symptoms*); Hypnotism (*Therapeutic Uses*); Memory in Health and Disease (*In Disease, Defects due to Non-Revival*).

Grangore. *See* Syphilis (*Historical*).

Granular Kidney. *See* Nephritis (*Renal Cirrhosis*).

Granulation. *See* Wounds (*Healing by Granulation or Second Intention*).

Granules.—Small particles found in the cells or fluids of the body; also, minute pills or globules.

Granuloma.—A vascular tumour consisting of granulation tissue or tissue resembling it; it may be infective in origin and nature (*see Actinomycosis*) or of unknown nature (*see* Pudenda, Ulcerating Granuloma of).

Grape Cure. *See* Therapeutics, Health Resorts (*Germany and Austria, Meran*).

Graphospasm.—Writers' cramp. *See* Neuroses, Occupation (*Writing Neurosis*).

Grass Bacillus. *See* Tuberculosis (*Diagnosis of the Tubercle Bacillus*).

Grasse. *See* Therapeutics, Health Resorts (*Riviera*).

Gravel. *See* Bladder, Injuries and Diseases (*Calculus*); Kidney, Surgical Affections (*Stone*); Urine, Pathological Changes in (*Phosphaturia, Oxaluria, Uric Acid, Urinary Calculi and Sediments*).

Graves' Disease. *See* Thyroid Gland, Medical (*Exophthalmic Goitre*); Thyroid Gland, Surgery of (*Graves' Disease*). *See also* Acromegaly (*Combination*); Glycosuria (*Forms, Alimentary*); Nose, Nasal Neuroses (*in Graves' Disease*); Pruritus (*Causation*); Rheumatism, Acute (*Associations*); Retina and Optic Nerve (*Enlargement of Retinal Vessels in Graves' Disease*); Thymus Gland (*Function*).

Gravidity (Graviditas).—Pregnancy (*q.v.*).

Gravity. *See* Physiology, Circulation (*Capillary Pressure, Effect of Gravity*).

Greece. *See* Balneology (*Greece*).

Green-Blindness. *See* Colour Vision (*Congenital Colour-Blindness, Varieties*).

Green Sickness. *See* Chlorosis.

Greenstick Fracture. *See* Fractures (*Radius and Ulna*).

Gregarines. *See* Micro-organisms (*Protozoa*).

Gregory's Mixture.—Pulvis Rhei Compositus. *See* Rhubarb.

"Greville" Method. *See* Hydropathy (*Hot Air Application*).

Grey Powder. Hydrargyrum cum cretâ. *See* Mercury.

Griffith's Mixture.—Mistura Ferri Composita. *See* Ferrum (*Ferri Sulphas*).

Grilling. *See* Invalid Feeding (*General Preparation of Meats*).

Grindelia.—The leaves and flowering tops of two species of the order Compositæ (G. robusta and G. squarrosa) which are official in the British Colonies (Indian and Colonial Addendum (1900) to the British Pharmacopœia of 1898); the *Extractum Grindeliæ Liquidum* is used, in doses of 10 to 20 m., as a stomachic, expectorant, and antispasmodic in asthma, bronchitis, and whooping cough. Externally grindelia has been employed in burns, vaginitis, gonorrhœa, and dermatitis (due to the use of *Rhus toxicodendron*).

Grindelwald. *See* Therapeutics, Health Resorts (*Switzerland*).

Grinders' Rot. *See* Lungs, Pneumonokoniosis (*Dust of Inorganic Origin*).

Grippe, La. *See* Influenza (*Historical*).

Grocco's Triangle.—A triangular paravertebral area of dullness found in cases of pleurisy on the opposite side to that on which there is the pleural effusion; it is not due to a second effusion.

Groin, The.

See also Hip-Joint, Injuries of; Hip-Joint, Diseases of; Hernia, Inguinal, Femoral; Scrotum and Testicle, Diseases of (*Hydrocele*); etc.

There is no region of the body in which a greater variety of lesions is to be met with than the groin, and here, if anywhere, a precise anatomical knowledge is of the greatest importance for diagnosis. The groin is not, like the axilla, a very definite anatomical region, but it may be regarded as forming the junction of the abdomen, pelvis, and thigh; and as being the area which includes Scarpa's triangle, Poupart's ligament, and the parts immediately adjacent.[1]

The skin of the groin is thin and delicate in texture, and the effects of abnormal distension, such as phlegmasia alba dolens, are shown by the presence of striæ.

Owing to the thinness of the skin the groin is a region selected for the inunction of drugs, *e.g.* mercurial ointments and cod-liver oil.

I. Anatomy of the Groin

The bony landmarks to be recognised are the anterior superior spine of the ilium, the anterior fifth of the crest of the ilium, and the spine and crest of the pubes. In thin individuals, and in those who have become emaciated, additional bony parts can be made out, such as the anterior inferior spine of the ilium, the portion of bone between it and the anterior superior spine, the upper part of the rim of the acetabulum, the head of the femur, especially on flexion and rotation outwards of the thigh, and part of the horizontal ramus of the pubes. Poupart's ligament is the lower border of the aponeurosis of the external oblique muscle, and extends, with its convexity downwards, from the anterior superior spine of the ilium to the spine of the pubes; its reflection on to the pectinal line is known as Gimbernat's ligament. Poupart's ligament is relaxed when the thigh is flexed, adducted, and rotated inwards, and this fact should be borne in mind when the groin is being palpated. Between Poupart's ligament and the innominate bone are the structures which pass from the abdomen to the thigh, viz. psoas, iliacus, and pectineus muscles; the femoral artery and vein; and the external cutaneous, the anterior crural, and the crural branch of the genito-crural nerves. The inguinal canal, which contains the spermatic cord in the male and the round ligament of the uterus in the female, is about an inch and a half in length, has a direction downwards and inwards, and is parallel to and a little above Poupart's ligament; it commences at the internal abdominal ring, which is half an inch above the middle of Poupart's ligament, and ends at the external ring, which lies immediately above, and internal to the spine of the pubes; accordingly, the neck of an inguinal hernia lies internal to the spine of the pubes, while the neck of a femoral hernia lies external to this spine. The superficial fascia of the lower part of the abdomen and the upper part of the thigh is divisible into two layers, between which lie the superficial vessels and nerves. The superficial layer of this superficial fascia is thick and fatty, and in the male is continued over the penis and the outer surface of the cord to the scrotum, where it helps to form the dartos. The superficial vessels which lie between the two layers of the superficial fascia are the principal ones which are divided in herniotomy. The deep layer of the superficial fascia is a thin fibrous layer, and lies upon the fascia lata to which it is adherent at the lower margin of Poupart's ligament. Extravasated urine may pass from the scrotum to the abdomen between the attachment of the deep layer of the superficial fascia to the symphysis pubis and the pubic spine. The saphenous opening in the fascia lata lies an inch and a half below and external to the pubic spine, and is covered by the cribriform fascia, which is regarded by some anatomists as being a part of the deep layer of the superficial fascia. The deep fascia, or the fascia lata, forms a complete investment for the thigh, and its attachments in the groin are to the crest of the ilium, Poupart's ligament, the body of the pubes, and the descending ramus of the pubes; its iliac portion is that part of the fascia which lies external to the saphenous opening, at the lower margin of which it is continuous with the pubic portion. The crural or femoral sheath, a fascial arrangement which invests the femoral vessels as they pass below Poupart's ligament, consists in front of a continuation downwards of the fascia transversalis, and behind of the fascia iliacus. In addition, it is covered in front by the iliac portion of the fascia lata, and the pubic portion of the fascia lata lies behind it. The sheath is funnel-shaped, being broader above than below; it is divided into three compartments by two thin septa; the femoral artery occupies the outermost compartment, the femoral vein lies in the middle, and the innermost compartment—known as the

[1] The general information which is given in this article will be supplemented in special articles, such as those upon "Hernia," "Aneurysm," etc.

femoral or crural canal—contains lymphatic vessels and a gland, and is the route which a femoral hernia takes in its journey from the abdomen to the thigh.

The lymphatic glands in the region of the groin are numerous and important, for when they are enlarged, as they often are, as the result of conditions about to be mentioned, they increase the difficulties attending the diagnosis of tumours in this region. The following is Sir Frederick Treves' lucid description of them :—

"They are divided into a superficial and deep set. The superficial set, averaging from ten to fifteen glands, is arranged in two clusters, one parallel and close to Poupart's ligament (the horizontal series), the other parallel and close to the long saphenous vein (the vertical series). The deep set, about four in number, is placed along the femoral vein, and occupies the crural canal.

"The inguinal glands receive the following lymphatics :—

"*Superficial vessels of lower limb* = vertical set of superficial glands.

"*Superficial vessels of lower half of abdomen* = middle glands of horizontal set.

"*Superficial vessels from outer surface of buttock* = external glands of horizontal set.

"*From inner surface of buttock* = internal glands of horizontal set (a few of these vessels go to the vertical glands).

"*Superficial vessels from external genitals* = horizontal glands, some few going to vertical set.

"*Superficial vessels of perineum* = vertical set.

"*Deep lymphatics of lower limb* = deep set of glands.

"The lymphatics that accompany the obturator, gluteal, and sciatic arteries, and the deep vessels of the penis, pass to the pelvis and have no concern with the inguinal glands."

Scarpa's triangle, which forms the lower portion of the groin, has for its base Poupart's ligament; its outer side, the sartorius muscle; its inner side, the inner border of the adductor longus; and its chief contents are the femoral artery and vein with their branches, including the origin of the profunda femoris artery and vein, the anterior crural nerve, and the termination of the internal saphenous vein.

Flexion of the thigh is a common condition, which may be temporary or permanent, partial or complete. In some cases it may be regarded as being a symptom and in others the result of disease. Though it will be fully dealt with in the article on "Diseases of the Hip-Joint," it may be mentioned in this place that temporary flexion of the thigh is often a symptom of value in the diagnosis of some of the diseases of the groin. It may be regarded as being Nature's method of diminishing pain about the hip-joint. Flexion of the thigh, especially when combined with adduction and rotation inwards, relaxes the fascia lata, Poupart's ligament, and the abdominal muscles and fascia, and in consequence the pain of such conditions as synovites of the hip-joint and strangulated inguinal or femoral hernia is relieved by reducing the pressure and allowing more room for the swelling. Extension of a flexed hip-joint should be carried out with caution, for cases are on record in which, during the operation, the skin and subcutaneous tissues were ruptured.

II. Diseases of the Groin

Skin diseases, as they affect the groin, merit no special attention; it may be noted, however, that pruritus genitalium not infrequently spreads from the scrotum or the labia to the groin, especially in very stout individuals. For further particulars about this and other affections of the skin the articles dealing with those subjects may be consulted.

Affections of the Lymphatic Glands.—Enlargement of the inguinal lymphatic glands is of frequent occurrence, the commonest causes being sepsis, tuberculosis, and syphilis. In most people, especially in males, and even in infants, lymphatic glands of the horizontal series are often found to be enlarged, as the result of frequent and trivial attacks of balanitis, or in consequence of the irritation of smegma preputii.

1. Septic inflammation of the glands is due, in the first instance, to the presence of a septic area in one of the regions—noted above—from which lymphatic vessels pass to one of the sets of lymphatic glands in the groin. Lymphangitis, of necessity, precedes this condition; its symptoms are—when the superficial lymphatics are affected—the presence of tender and somewhat cord-like, minute, red streaks in the situation of the lymphatic vessels, accompanied by some cellulitis of the adjacent tissues, which have an appearance almost indistinguishable from erysipelas. "Phlebitis closely resembles lymphangitis in its symptoms; a thrombosed vein forms a deeper-seated coarser cord than a similarly affected lymph vessel, the cutaneous redness is not so vivid, the pain is less acute, the general fever is not so intense, and the tendency to glandular involvement is less." Inflammation of the deep lymphatics is not easily differentiated from ordinary cellulitis.

The septic area from which the lymphatic vessels pass should be carefully sought for—a point which is often overlooked—and treated on general principles. If this lesion is detected early and efficiently treated, suppuration in the lymphatic glands may be prevented. If, however, suppuration has occurred, the glands should be laid freely open and stuffed with sterilised gauze. More harm than good may be done by making too small an incision in the gland, and the timid surgeon is apt to be over-cautious when operating upon glands in close proximity to large and important blood-vessels. The incision

should be parallel to the principal vessels and nerves. Cellulitis of the surrounding tissues occasionally accompanies suppuration in the glands, and, if free incision of the glands does not arrest or cure the cellulitis, it is advisable to prolong the incision into the inflamed area. One of the commonest forms of suppuration in the inguinal glands is the result of a soft chancre on the penis, the treatment of which differs in no respect from that just mentioned. Before suppuration has occurred great relief may be given to the patient by applying fomentations of hot water, or hot lead and opium lotion; frequent changing of the fomentations enhances the value of this treatment.

2. A tuberculous condition of the inguinal lymphatic glands is not uncommon, resulting either as an infection from a tuberculous area, or as part of a general tuberculous infection of the glands. Several of the glands are usually affected, and they may remain enlarged for some time, and then disappear without any local treatment; or they may remain enlarged for a lengthened period without giving rise to any trouble; or they may undergo caseous degeneration, in which case the glands should be excised, or thoroughly curetted; or suppuration may take place in them, in which case incision being practically impossible or difficult to perform, curetting should be resorted to. General treatment, in the form of cod-liver oil, fresh air, and sunlight, should not be neglected.

3. Syphilitic affection of the inguinal glands is frequently met with, usually as a sequel to a hard chancre of the penis. Inguinal glands, when enlarged from this cause, do not, as a rule, suppurate if the patient rest; but if they do, a free incision and the application of some mercurial dressing, combined with constitutional treatment, is the indication.

In Hodgkin's disease or lymphadenoma, the lymphatic glands in the groin are enlarged, as are the lymphatic glands throughout the body generally. The glands form irregular and nodulated masses of various sizes, are either soft or firm, usually painless, and at first freely movable; later they may become adherent, but rarely caseate or suppurate. In the majority of cases no local treatment in the groin is necessary; for the general treatment of this condition the article on Hodgkin's disease should be consulted. (The diagnosis of this condition presents little difficulty; enlargement of the lymphatic glands, especially in the neck, the axilla, and the groin, associated with anæmia, is characteristic.)

Elephantiasis may be mentioned, for in those cases in which the leg and the thigh are affected, the groin shares in the general hypertrophy of the skin and subcutaneous tissues. Fistulæ in the groin are occasionally met with in this condition as the result of rupture of obstructed and distended lymphatic vessels.

Phlegmasia alba dolens, which is due to thrombosis of the femoral vein—and probably also of the iliac veins—produces extensive œdema of the lower extremity, resembles a mild case of elephantiasis in appearance, but could scarcely be mistaken for that condition.

The following is a list of the conditions which may be met with in the groin, each of which will be described in more detail in special articles (*q.v.*)—

Abscess and cellulitis; aneurysm; aneurysm with suppurating sac; arterio-venous aneurysm; varix of long saphenous vein; varix of femoral vein; phlebitis; phlegmasia alba dolens; enlargement of glands (adenitis; bubo); lymphadenoma; hernia,—inguinal, femoral, and obturator, with their varieties,—reducible, irreducible, and strangulated,—also the morbid conditions of the sac occasionally met with, viz.: hydrocele of the sac, and accumulation of ascitic fluid in the sac; various tumours—simple, sarcomatous, carcinomatous, and vascular; bursal tumours and cysts; undescended ovary and testicle, which may become inflamed in the inguinal canal; hydrocele, simple or diffuse, of the cord, and hæmatocele of the cord; psoas and iliac abscess; perityphlitic and perinephritic abscess; hip-joint disease; synovitis and suppuration in the hip-joint; dislocation, congenital and acquired, of the hip-joint; rider's bone, attributed to ossification of the tendon of the adductor longus or magnus as a consequence of injury.

The space allotted to this article prevents an elaborate description being given of the differential diagnosis of the numerous conditions just mentioned, and, therefore, it must suffice if a short account be given of the procedure to be adopted, and the points to be borne in mind, in making a diagnosis of the nature of a lump, swelling, or tumour in the groin.

1. The Age of the Patient.—Such conditions as aneurysm, malignant tumours, and rider's bone are unlikely to be met with in the very young, while in the aged tuberculous lesions are rare.

2. History of the Case.—A history of injury should be carefully inquired into; accurate information should be obtained about former illnesses and injuries and previous operations. The sudden appearance of the swelling would eliminate the different varieties of tumour, but might suggest hernia or aneurysm. The rapidity in growth of the swelling may vary between wide limits—a tumour, almost stationary as regards size, might be a hydrocele or one of the simple tumours, whereas rapid increase in growth might point to malignant disease, abscess, aneurysm, etc.

3. The Character of the Swelling.—If it disappears on manipulation it is unlikely to be anything but a hernia or a congenital hydrocele; if it is much reduced in size on pressure or by position it may be a varix, or a psoas, or iliac abscess. It is certain to be a varix if pressure

above increases its size. Resonance on percussion would suggest an enterocele. The presence of fluctuation would eliminate a large class of swellings, *e.g.* the tumours proper, etc., but would indicate glandular and simple abscess and rarely aneurysm. Impulse on coughing is present in reducible and irreducible hernia, psoas abscess, and varix, and occasionally in cysts. A pulsating tumour is probably an aneurysm, or a vascular malignant growth, possibly a swelling, fluid or even solid, with pulsation communicated from the femoral artery. The presence of a thrill is characteristic of an aneurysm, or more rarely of a fluid tumour lying on the femoral artery. Pain is a prominent symptom in inflammatory conditions, in neuroma, and in tumours which press upon sensory nerves.

4. Signs of inflammation should also be noted. The swelling may be primarily an inflammatory one, *e.g.* abscess; or a pre-existing tumour, *e.g.* an aneurysm may become inflamed.

5. It is important to ascertain the existence of morbid conditions in any other part of the body which may have an important bearing on the lesion in the groin. Abdominal symptoms are, of course, a prominent feature in strangulated hernia and perityphlitic abscess, and their occurrence should be carefully noted. Thus, enlarged lymphatic glands in the neck and axilla, as well as in the groin, associated with anæmia, would confirm a diagnosis of Hodgkin's disease, and similarly a diagnosis of tuberculosis or syphilis might be made.

6. In the examination of the tumour it should be carefully ascertained if the tumour in the groin is confined to the groin, or if it extends into another region. Attention to this point might confirm a diagnosis of, *e.g.* psoas abscess.

III. Injuries of the Groin

Dislocations of the femur and fractures of the femur and of the pelvis are described in articles devoted to these subjects, and the possibility of suppuration following separation of the upper epiphysis of the femur should be borne in mind. Gunshot wounds are, especially during warfare, very common, and the nature and extent of the injury depends upon the size and shape of the projectile—an expanding bullet inflicting a very much more serious injury both upon the tissues and the bones than a non-expanding bullet; upon the velocity of the projectile—the greater the velocity the less serious the injury; and upon the distance which the projectile has to travel. Full details on these points will be found in the articles on "Gunshot Wounds." (*See* Ankle-Joint, Region of, Injuries; Knee-Joint, Injuries of; Œsophagus, Wounds of.) When the artery is injured a traumatic aneurysm may result. Damage to the vein may cause an arterio-venous aneurysm, and if nerves, such as the anterior crural, are divided paralysis will result when the motor filaments are implicated, anæsthesia when the sensory filaments are implicated. The bone may escape injury, or it may be shattered. Every attempt must be made to save the limb, for few operations are attended with a higher mortality than amputation at the hip-joint, which such an injury would necessitate. Stabs and other punctured wounds may produce injuries similar to those just described, with the reservation that the subjacent bones can be, and usually are, damaged to only a slight extent.

Burns of the groin, more especially if deep, should be carefully treated, for unless the thigh is maintained extended by splints or extension during the healing process a cicatrix may result, causing almost incurable flexion of the thigh.

Great care should be taken to prevent the suppuration of wounds in the groin, for owing to the superficial position of the main vessels deep ulceration may result in secondary hæmorrhage.

Ground Air. *See* Meteorology (*Barometer, Atmospheric Pressure*).

Ground Itch. *See* Skin-Diseases of the Tropics (*Due to Animal Parasites, Pani Ghao*).

Ground Water. *See* Diphtheria (*Etiology, Level of Ground Water*).

Growing Pains. *See* Heart, Myocardium and Endocardium (*Etiology, Rheumatism*); Hip-Joint, Injuries of (*Coxa Vara, Growing Pains*).

Growth. *See* Children, Development and Clinical Examination of (*Growth in Weight and Length*).

Growth-Fever. *See* Bones, Diseases (*Pyogenic Diseases, Growth-Fever*).

Gruber-Widal Reaction. *See* Immunity (*Typhoid, Gruber-Widal Reaction*).

Grübler's Peptone. *See* Physiology, Food and Digestion (*Absorption of Food, Mode*).

Gruebler's Tumour.—A swelling over the wrist in cases of lead-poisoning, in which there is paralysis of the ante-brachial type (paralysis of the extensors of the fingers and wrist); the swelling is due to slight displacement backward of the bones of the carpus.

Gruel. *See* Invalid Feeding (*Prepared Foods, Diet during Convalescence*).

Guagno. *See* Balneology (*France, Corsica, Sulphur*).

Guaiaci Lignum.—Lignum vitæ; the heart wood of *Guaiacum officinale*, or of *Guaiacum sanctum*, found in the West Indies. Its chief constituent is Guaiac resin. It is contained in *Liquor Sarsæ Compositus Concentratus.*

Guaiaci Resina.—Obtained from the stem of *Guaiacum officinale* or *Guaiacum sanctum*. It occurs in large masses of a greenish brown colour and a balsamic odour. It contains three resins—Guaiaconic acid, Guaiac acid, and Guaiaretic acid. It is soluble in alcohol, ether, and chloroform. *Dose*—5-15 grs. *Preparations*—1. Mistura guaiaci. *Dose*—½-1 ℥. 2. Tinctura guaiaci ammoniata. *Dose*—½-1 ʒ. 3. Trochiscus guaiaci resinæ. 3 grs. in each. Guaiac is contained in Pilula hydrargyri subchloridi composita.

Guaiac resin is very disagreeable to take, and its therapeutic value being somewhat doubtful, it is now not very often prescribed. It is still, however, a favourite with some in chronic rheumatism and lumbago. It has a slight purgative action, and is commonly given to gouty patients along with some other laxative in the form of a pill. The "Chelsea Pensioner" contains guaiac, sulphur, magnesium carbonate, ginger, and treacle. Guaiac has a beneficial action in some forms of chronic sore throat.

Guaiacol.—Guaiacol, in its impure form, is a colourless liquid obtained by the distillation of guaiac resin or beech creosote; pure guaiacol is a monomethyl ether of pyrocatechin ($C_7H_8O_2$), and is a crystalline substance; the dose of the former is 1 to 5 m. (in capsules, diluted with oil), and that of the latter is 1 to 5 grains (in cachets); it is an antiseptic, and has been widely employed in the treatment of pulmonary consumption; it is not official.

Guaiacum Test. *See* MEDICINE, FORENSIC (*Examination for Blood Stains, Guaiacum Test*); HÆMATURIA.

Guaiamar.—An antiseptic, used sometimes in pulmonary consumption; it is guaiacol-glycerylether ($C_{10}H_{14}O_4$); the dose is 5 to 20 grains.

Guaiaquin.—Guaiaquin is quinine guaiacol bisulphonate ($C_6H_4O_2CH_3HSO_3 \cdot C_{20}H_{24}N_2O_2$). It is believed to combine the pharmacological actions of guaiacol (antiseptic) and of quinine (antiperiodic). It has been used in doses of 1 to 5 grains in enteritis.

Guanidin. — An extractive substance (CN_3H_5) nearly allied to urea. *See* PHYSIOLOGY, TISSUES (*Muscle, Chemistry of*).

Guanin. — Guanin, or imidoxanthin ($C_5H_5N_5O$), or amino-oxypurin, is found in guano, and sometimes in the human body (liver, spleen, etc.). *See* GOUT (*Gout in Lower Animals*); LIVER, PHYSIOLOGY OF.

Guarana.—Guarana or Brazilian cocoa consists of the seeds of *Paullinia cupana*, powdered and made into a paste; it contains guaranine or caffeine ($C_8H_{10}N_4O_2 \cdot H_2O$); the dose of guarana is 20 to 60 grains, and of the *Elixir Guaranæ*, ½ to 2 fl. dr. It is used chiefly in cases of megrim headache; it is not official.

Gubernaculum Testis. *See* SCROTUM AND TESTICLE, DISEASES OF (*Diseases of Testicle and Cord, Development of Testicle*).

Guerin, Valve of. *See* URETHRA, DISEASES OF (*Anatomy of Male Urethra*).

Guillotine.—An instrument for the rapid removal of prominent parts, such as enlarged tonsils. *See* TONSILS, DISEASES OF (*Hypertrophy, Treatment*).

Guinea Worm. *See* FILARIASIS (*Filaria Medinensis*).

Gull's Disease.—Myxœdema as it is met with in adults. *See* THYROID GLAND, MEDICAL (*Myxœdema*).

Gully Trap. *See* SEWAGE AND DRAINAGE (*Traps*).

Gum Acacia.—Acaciæ gummi is obtained from various species of acacia, plants belonging to the Leguminosæ. It has the form of colourless round or ovoid tears; it contains *arabin* or *arabic acid* ($C_6H_{10}O_5$), and is often mixed with such impurities as starch and gum-resins. The *Mucilago Acaciæ* is given in doses of 1 to 4 fl. dr., and the gum itself is contained in all the trochisci and in some pills; it acts as a demulcent, and is used, in prescribing, for the making of emulsions. *See also* DEMULCENTS.

Gumboil. *See* TEETH (*Diseases of Peridental Membrane*).

Gumma. *See* SYPHILIS (*General Pathology, Tertiary Syphilis*, etc.); BONE, DISEASES OF (*Syphilitic*); CHOROID, DISEASES OF (*Gumma*); CONJUNCTIVA, DISEASES OF (*Syphilis*); JOINTS, DISEASES OF (*Syphilitic, Gummata*); KNEE-JOINT, DISEASES OF (*Tuberculous Disease, Diagnosis*); LIVER, DISEASES OF (*Syphilis, Tertiary*); LUNGS, SYPHILIS OF; MEDIASTINUM (*Tumours, Gummata*); MUSCLES, DISEASES OF THE (*Inflammatory, Syphilitic, Gummata*); PHARYNX, CHRONIC INFECTIVE DISEASES (*Syphilis*).

Gummi Indicum.—A preparation closely resembling gum acacia; it is an exudation from the wood of Anogcissus latifolia; it is official in the Colonies and India; and the only preparation is the *Mucilago Gummi Indici*. *See* GUM ACACIA; DEMULCENTS.

INDEX

END OF VOL. III

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